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DISEASES *of the* CHEST

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# Postmortem Pneumothorax*

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If the thorax of a cadaver is opened, or if a hypodermic needle is inserted post mortem through the chest wall into the pleural cavity, there is produced a small pneumothorax space. The room air enters the pleural cavity until the intrapleural pressure becomes atmospheric. The size of the space so produced depends chiefly upon the degree of retractility of the lung; and secondarily upon such factors as the nature of the disease in the lung, the degree of subatmospheric pressure in the pleural space, the presence of pleural adhesions, the mobility of the chest cage, and the length of time after death. Under the most favorable conditions the pneumothorax occupies from 5 per cent to 15 per cent of the pleural cavity.

We have increased the size of this pneumothorax by pumping air under pressure into the pleural cavity. Large pneumothorax spaces were thus produced and a variety of pulmonary collapses obtained. Of course, such a positive pressure pneumothorax over a dead and partially inelastic lung cannot be compared too closely with a subatmospheric pneumothorax over a living lung, but many of the effects are so similar and even identical that it stresses the importance of the strictly mechanical factors involved in pneumothorax.

## *Technique*

Shortly after death, from a few minutes up to three hours, the cadaver was brought to the x-ray room and seated in a specially constructed chair mounted on wheels and having a cassette holder in its back.[1] Our room was so arranged that the cadaver could be

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placed in front of the upright fluoroscope or the regular x-ray tube to permit fluoroscopy and the taking of a six-foot film without disturbing the position of the chair or the cadaver.

A six-foot film was made of the thorax prior to any procedure. Then, under fluoroscopic guidance, a 16-gauge needle was inserted through the chest wall into the pleural cavity. The needle was attached to a Potain aspirator and air was pumped into the pleural cavity. The pneumothorax so produced was followed under constant fluoroscopic vision. The pressure used was often considerable and was measured by an attached manometer. Air was forced into the pleural cavity until the lung ceased to collapse. At this point of maximum collapse there was usually an escape of pleural air around the needle into the muscle planes and subcutaneous tissue (Figures 3, 5, 8, 17).

The final intrapleural pressure (after the maximum pulmonary collapse) ranged from atmospheric to 30 cm. water. The lung remained small, but its size could be altered by either withdrawing some of the pleural air or by producing a contralateral pneumothorax. If even a slight amount of air was withdrawn the intrapleural pressure became subatmospheric and so remained.

In this study, pneumothorax was produced from several minutes to three hours after death, in bodies still warm, in some that were at room temperature, and in others in complete rigor. The degree and type of pulmonary collapse was not materially altered by these factors during this time interval.

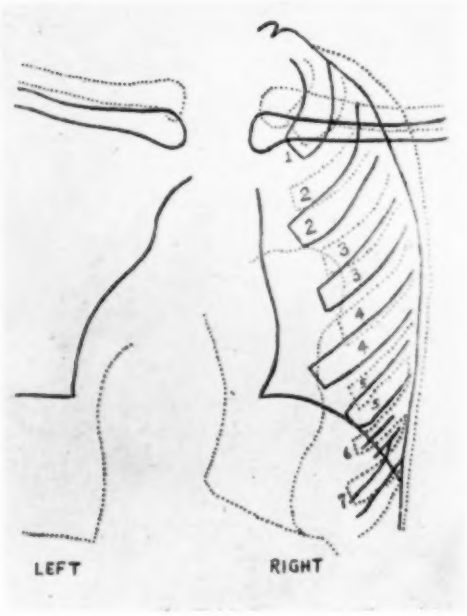


FIGURE 1

Effect of a postmortem pneumothorax on the motion of the bony thorax. The solid line shows the ribs, clavicle, heart, and diaphragm prior to the pneumothorax. The dotted line shows them and the right lung after a bilateral collapse.

### *Material*

This study was begun in 1937 and ended in 1941. A total of 80 cadavers was used. The chief pulmonary lesion was cavernous tuberculosis in 74 cases, miliary tuberculosis in 2 cases, emphysema in 2 cases, silicosis in one case, and bronchiogenic carcinoma in one case. From a total of 160 sides, some degree of pneumothorax was obtained in 119 (or 74 per cent). The other 41 sides showed complete obliteration of the pleural space. Necropsies were done in 34 cases (or 42 per cent).

The results obtained will be discussed in sections dealing with the effects of the pneumothorax upon the bony thorax, diaphragm, heart, anterior mediastinum, lungs, and pleural cavities. The effects of aspiration of air from pulmonary cavities will also be mentioned. In general, all these structures were profoundly affected and showed many changes similar to those seen with pneumothorax in the living patient.

### *Bony Thorax*

In both uniform and irregular pneumothoraces the ribs were elevated and became more horizontal. This movement upward was greatest in the lower ribs anteriorly and least in the upper ribs posteriorly. The movement upward measured from 1 to 3 cm. anteriorly. The interspaces were widened, especially the lower ones, and the sternum and clavicles were also elevated though

FIGURE 2

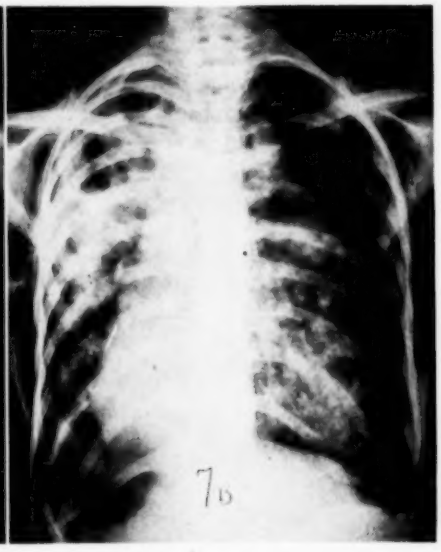


FIGURE 3

*Figure 2, Case 7:* Postmortem film prior to pneumothorax. The right apex shows good aeration.—*Figure 3, Case 7:* Right pneumothorax with "atelectasis" in upper lobe, selective collapse, air in soft tissues, and displacement of the heart.

to a lesser degree than the ribs. The width of the hemithorax with the air space showed an increase in size by about 2 cm. The effects were similar to those seen with pneumothorax in the living (Fig. 1).

*Diaphragm*

During the induction of the pneumothorax the hemidiaphragm moved downward and continued to do so until a maximum pulmonary collapse was obtained. The descent was very slight with small and irregular pneumothoraces, but reached from 4 to 10 cm. in large collapses. A small basal pneumothorax produced a greater descent than a large apical one. Each diaphragmatic leaf moved independently of the other and maintained its dome-shaped contour. Despite the many poor collapses in this series an appreciable descent occurred in 95 sides, or 80 per cent (Figs. 5, 7, 8, 12, 14).

*Heart*

In this group of 119 pneumothoraces the heart and large vessels were fixed and immobile in 30 cases and freely movable in 89 cases (or 75 per cent). In all cases the prepneumothorax picture was typically cadaveric—wide heart and vessel shadows and a small longitudinal diameter. The cases showing a mobile heart presented some degree of movement in two directions. The heart

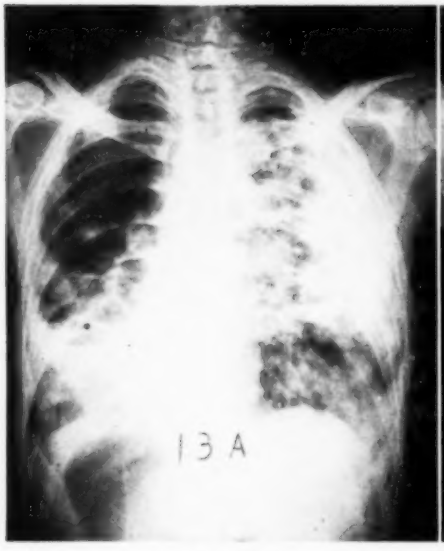


FIGURE 4

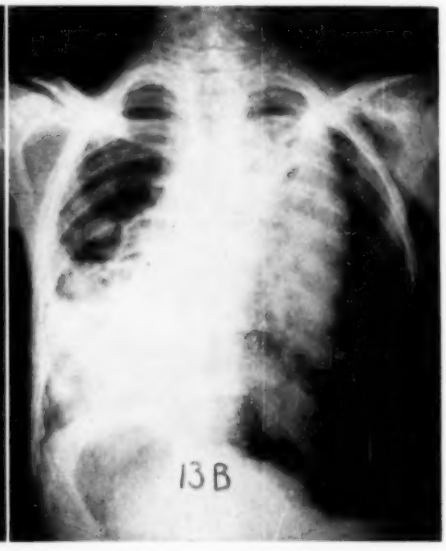


FIGURE 5

*Figure 4, Case 13:* Postmortem film prior to pneumothorax. The left lung is completely excavated; the right lung shows two large cavities.—*Figure 5, Case 13:* Right pneumothorax. Note the "airlessness" in the upper two-thirds of the right lung, the obscuration and small size of the two large cavities, the descent of the right henidiaphragm, the cardiac displacement, and some intrapleural fluid.

and vessels were lengthened (sometimes considerably) and the transverse diameter was decreased. The antero-posterior diameter was increased so that the heart was flatter when viewed from the side and occupied more space posteriorly. The effect of these two movements presented a heart which was lengthened from its base to its apex, flattened, and showed some increase in size posteriorly.

A right pneumothorax displaced the heart to the left. The shift was often considerable and reached 6 to 8 cm. The right border of the heart was often not seen because of its displacement. A left pneumothorax displaced the heart similarly to the right. A unilateral pneumothorax produced little or no lengthening of the heart. In bilateral pneumothorax the heart was first displaced to one side and then back to the midline by the second pneumothorax, and its final picture showed it to be longer and narrower than before. Only a bilateral pneumothorax and a bilateral descent of the hemidiaphragms produced much lengthening of the heart. In such cases the heart assumed the typical picture seen in advanced emphysema in the living—long and narrow with often a prominent pulmonary conus.

In several cases of right-sided pneumothorax the first displacement of the heart occurred in the upper left border producing a bulge and a deformity similar to that seen in mitral stenosis (Figs. 1, 3, 5, 7, 8, 10, 12, 14, 17).

### *Anterior Mediastinal Hernia*

A pleural anterior transmediastinal hernia occurred in some degree in a large number of the pneumothoraces but was especially marked in 16 cases. The mesial pleural reflection and the contained pleural air was easily followed fluoroscopically across the midline to the other side. These 16 cases showed a pleural shift of 2 to 6 cm. past the mid-sternal line and the herniation grew larger as the pneumothorax was increased. The hernial space extended from the first rib to the fourth and often to the fifth. In three cases, as the pneumothorax was increased, the greatest width of the herniation moved upward from the level of the fourth rib to that of the third.

The herniation was usually made smaller by a contralateral pneumothorax and, in cases of uniform bilateral collapse, was entirely abolished. Several cases showed small posterior mediastinal hernias (Figs. 7, 8, 13, 14).

### *Type of Pulmonary Collapse*

A unilateral pneumothorax was obtained in 41 cases and a bilateral one in 39 cases resulting in 119 spaces. In 9 spaces the collapse

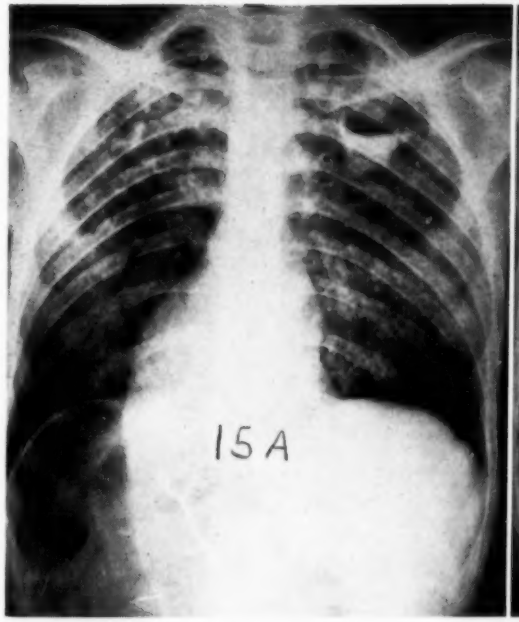


FIGURE 6

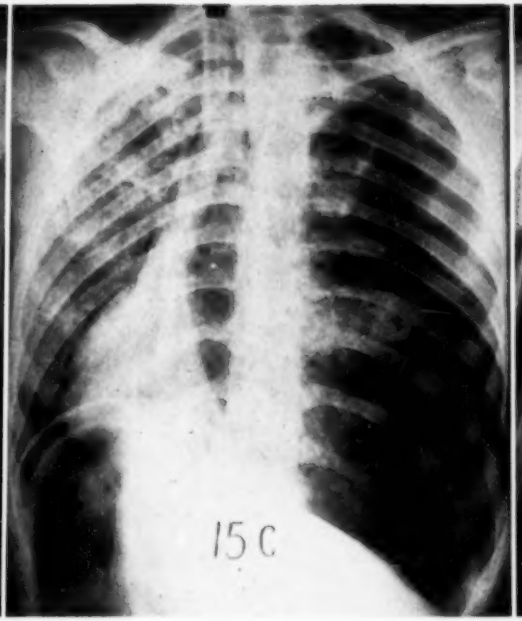


FIGURE 7

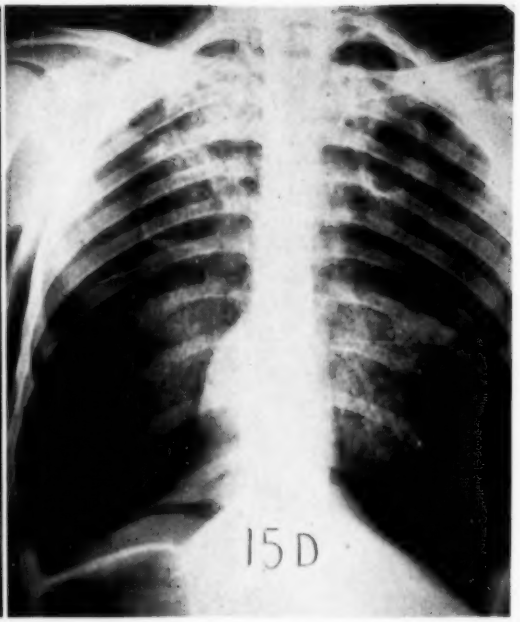


FIGURE 8

*Figure 6, Case 15:* Postmortem film prior to pneumothorax.—*Figure 7, Case 15:* Right pneumothorax. Note the depression of the right hemidiaphragm, displaced heart, anterior mediastinal hernia, distortion of the pulmonary cavity.—*Figure 8, Case 15:* Bilateral pneumothorax. Note the low diaphragm, cardiac position and contour, abolition of the hernia.

was small, localized, and insignificant. In 76 spaces the pneumothorax was modified greatly by pleuro-pulmonary adhesions and presented all degrees and types of collapse. In 34 sides the air was quite uniformly distributed over the entire lung; 10 of these showed a moderate number of adhesions which apparently prevented a better local collapse, and 24 showed a more ideal collapse involving the entire lung and not affected by the few fine adhesions seen.

The 34 sides mentioned showed collapses varying from 20 to 80 per cent with an average collapse of 50 per cent for the group. One lung showed a 20 per cent collapse; 5 lungs a 20 to 30 per cent collapse; 9 lungs showed 30 to 40 per cent; 11 lungs showed 50 per cent; 3 lungs 60 per cent; 2 lungs 70 per cent; and 3 lungs 80 per cent.

The 76 sides mentioned above presented irregular collapses varying from a relatively small volume to over 50 per cent. Thus 50 lungs showed a collapse under 30 per cent; 22 lungs from 30 to 50 per cent; and 4 lungs over 50 per cent.

### *Pneumothorax in a Free Pleural Space*

The 24 sides with the more ideal collapse showed very few or no adhesions roentgenologically. The collapse obtained varied from 30 to 80 per cent with a mean average of 55 per cent. This group included 20 cases of ulcerative or exudative tuberculosis, 2 cases of miliary tuberculosis, 1 case of bronchiogenic carcinoma with emphysema, and 1 case of emphysema with myocardial failure.

Necropsies were done in 12 cases of this group and the adhesions noted. One case showed no adhesions at all; 7 cases showed only a few slight strands; and 4 cases a moderate number of long, string-like adhesions. None appeared to influence the degree of collapse as all were fine, long, and not under any tension. The case without any adhesions showed a collapse of 80 per cent; the 7 cases with the few strands showed a mean collapse of 53 per cent (40, 40, 40, 50, 60, 70, 70 per cent) and the 4 cases with more adhesions showed an average collapse of 52 per cent (40, 50, 50, 70 per cent).

The degree of pulmonary *infiltration* or the amount of disease present was divided roughly as mild, moderate, and extensive. Nine cases with a mild amount of disease showed an average collapse of 60 per cent. Six cases with a moderate amount showed an average collapse of 50 per cent; of this number there were three with localized disease and a collapse of 60 per cent, and three with uniformly scattered disease and a collapse of 40 per cent. Nine cases with extensive disease averaged 45 per cent collapse and, of this number, five with localized disease and four

with disseminated disease showed the same degree of collapse.

The degree of pulmonary *cavitation* and the degree of maximum collapse were correlated. Eleven lungs had no cavities and showed an average collapse of 60 per cent; eight lungs had cavities up to 4 cm. and showed a collapse of 50 per cent; two lungs with cavities from 4 to 6 cm. showed a collapse of 40 per cent; and three lungs with cavities over 6 cm. showed a mean collapse of 35 per cent.

The amount of *normal lung tissue* appeared to be a factor. Seven lungs showed only a slight amount of normal tissue and presented an average collapse of 45 per cent; six cases with a moderate amount of normal tissue showed a 45 per cent collapse; eight cases with a large amount of normal tissue showed a 70 per cent collapse. Three cases with emphysema showed a 55 per cent collapse.

In summary, (1) the greater the amount of pulmonary disease the less was the degree of collapse obtained in an essentially free pleural space; (2) the greater the degree of cavitation, the less was the collapse; (3) the more normal lung tissue present, the greater was the collapse, and (4) the three cases of emphysema showed less collapse than those with a similar amount of normal tissue. Therefore, one may expect the best pneumothorax collapse in instances showing a small amount of disease, a small cavity or none at all, a large amount of normal alveolar tissue, and no emphysema.

FIGURE 9

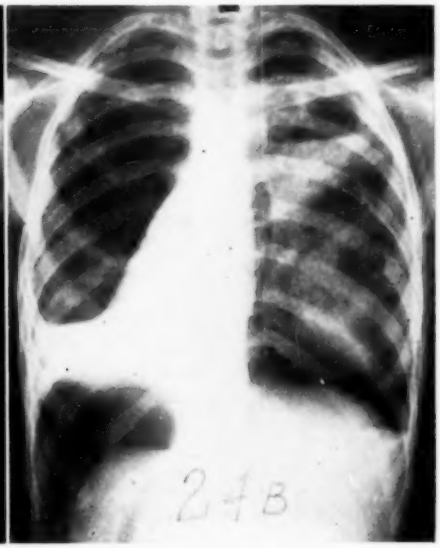


FIGURE 10

*Figure 9, Case 24:* Prior to pneumothorax. The left lung is completely excavated; the right lung shows a large apical cavity and a smaller one below it. *Figure 10, Case 24:* Right pneumothorax. The small cavity is "closed" and the large one is smaller and shows a greater fluid level. Note also the cardiac displacement and its effect on the fluid level in the left-sided cavity.

### *Selective Collapse*

A total of 14 pneumothoraces showed selective collapse; 10 at the apex and 4 at the base. Seven of this group were associated with a relatively free pleural space and 7 had gross adhesions which did not prevent the collapse. The diseased apical portion was seen fluoroscopically to undergo collapse first after the injection of only a small amount of air; the rest of the lung then followed. Two of these cases were associated with premortem lobar atelectasis (Figs. 3, 13, 17).

Four of these cases were necropsied. One showed extensive caseous pneumonia with small islets of normal tissue, little liquefaction, and normal bronchi grossly. The second showed extensive confluent caseous masses and normal bronchi. The third also showed confluent caseous masses, normal bronchi, and no gross cavitation. The fourth showed numerous exudative areas at the base and the bronchi were normal.

The ten cases not necropsied showed the following prepneumothorax roentgen pictures:

(1) dense exudative lesions in entire lung and chiefly in the lower lobe,
(2) dense exudative areas chiefly in the upper lobe (Figs. 2, 3),
(3) moderately dense productive nodules chiefly in the upper lobe,
(4) contracted, atelectatic, excavated, and fibroid upper lobe,
(5) (6) bilateral, fine, symmetrical miliary tuberculosis (Figs. 11, 13),
(7) moderately dense productive lesions in the right lower lobe with a 6 cm. cavity at the apex of this lobe,
(8) moderately dense infiltration chiefly in the upper lobe with a 3 cm. apical cavity,
(9) light exudative infiltration in the upper lobe,
(10) dense confluent bronchopneumonic areas at base and normal bronchi (Figs. 16, 17).

It is to be noted that 8 of these lungs showed extensive disease in the selectively collapsed lobe, 1 had a miliary tuberculosis, and 1 had a light infiltration.

### *Atelectasis*

The term atelectasis is used to denote airlessness which occurred only in a lobar distribution in this group of cases. Six such cases were noted and two of them were present prior to the pneumothorax. The right upper lobe was involved four times, the right lower lobe once, and the left lower lobe once. Only one case was necropsied. A description of each case follows:

FIGURE 11 FIGURE 12 FIGURE 13 FIGURE 14

*Figure 11, Case 27:* Postmortem film showing a fine miliary disseminated infiltration.—*Figure 12, Case 27:* Right pneumothorax showing a large pulmonary collapse, low hemidiaphragm, displaced heart, and anterior hernia.—*Figure 13, Case 27:* Left pneumothorax. Note collapse of the upper lobe only.—*Figure 14, Case 27:* Left pneumothorax continued. Note the pulmonary collapse, low diaphragm, shape and position of the heart, absence of the hernia, and change in shape of the bony thorax.

*Case 1* (Figs. 2, 3): Prior to collapse the x-ray film showed a dense exudative lesion in the entire lung especially marked at the right subapex. After the pneumothorax the lobe showed a selective collapse and the rest of the lung an ordinary one. The previously aerated apex became a dense homogeneous shadow. A bronchogram was then made with a barium emulsion and it showed poor filling of the larger bronchi and none of the smaller ones. Prior to the pneumothorax the postmortem intrapleural pressures were −13 cm. water over the upper lobe and −5 cm. over the lower lobe.

*Case 2* (Figs. 4, 5): The x-ray film showed a series of cavities occupying two-thirds of the entire lung field and measuring 17 cm., and a dense homogeneous shadow laterally over the right upper lobe. After pneumothorax the upper two-thirds of the lung showed a dense homogeneous "airless" picture through which rarefactions could be seen and the lower one-third showed the usual collapse. Bronchograms were then made and showed no bronchial filling in the upper lobe. Post-mortem intrapleural pressures taken prior to the pneumothorax were much more subatmosphreic on the right side than on the left side viz. taken at four comparable points the readings on the right side were −3, −9, −7, −3 cm. $H_2O$ and on the left side 0, −1, −2, 0 cm. $H_2O$.

*Case 3:* The x-ray film showed an airless, contracted right upper lobe. After pneumothorax the lobe showed a selective collapse, was smaller, the homogenous shadow was denser, and the cavities fainter and smaller. Bronchograms were then made and showed normal bronchi and an open cavity. The pre-pneumothorax post-mortem intrapleural pressure was −14 cm. $H_2O$ over the upper lobe and −10 cm. $H_2O$ over the lower lobe.

*Case 4:* The x-ray film showed the right lower lobe to be dark and hyperradiolucent and containing a moderate, scattered infiltration. After pneumothorax this lobe showed a 50 per cent collapse and a dense, homogeneous shadow. Bronchograms showed a moderate, saccular bronchiectasis in this lobe. Pre-pneumothorax pressures were −1 cm. $H_2O$ at the base and zero over the apex.

*Case 5:* The x-ray film showed a dense exudative infiltration in the right upper lung field with cavitation measuring 12 cm. After pneumothorax the upper two-thirds of the lung became a dense, homogeneous shadow through which the rarefactions could be seen faintly. Bronchograms revealed the cavities to be open but most of the bronchi did not fill. Pre-pneumothorax intrapleural pressures at four points on the right side were, −12, −10, −12, and −9 cm. $H_2O$ and on the left side −14, −2, −3, and −1 cm. $H_2O$.

*Case 6:* The x-ray film showed a dense homogeneous left lower lobe with a 10 cm. cavity. After pneumothorax the dense shadow became even denser but the cavity showed no change in size. Bronchograms showed no filling of the smaller bronchi. Necropsy showed dense, confluent, tuberculous, caseous masses, a 10 cm. cavity, small silicotic nodules, considerable fibrosis, tuberculous ulceration of the left lower lobe bronchus and its branches, and no air-containing lung.

Thus, atelectasis occurred in 4 cases of exudative disease, one case of productive disease, and one case of fibroid disease. In 2 cases the bronchi were open and in 4 cases diseased or poorly

filled. Emphysema occurred in one case. The intrapleural pressure was greater subatmospherically (more negative) in the five cases taken over the areas subsequently showing the "atelectasis."

*Alveolar Changes*

The collapsed lung post-mortem showed no roentgen changes differing greatly from that during life (Fig. 15). The lung showed collapse to a variable degree and contained air. Bronchograms showed good alveolar filling and necropsy examination revealed air in the alveoli. In some cases, the tracheobronchial tree was first filled with a barium emulsion and then the pneumothorax was begun. During the collapse, air bubbles could be seen moving from the parenchyma through the bronchi to the trachea and larynx.

*Cavity Changes*

The total number of cavities in this entire group was not counted but reached a considerable figure. However, 25 cavities showed a noteworthy change in size with the pneumothorax.

One cavity measuring 2 x 1 cm. was closed roentgenologically. It lay at the level of the seventh dorsal spine and was "closed" by a basal pneumothorax which went as high as the sixth dorsal spine (Figs. 9, 10). A large 11 cm. cavity just above it was only slightly changed by the pneumothorax. At necropsy the "closed" cavity showed approximation of its walls and the bronchi leading to it were normal and patent.

A gross decrease in size occurred in 24 cavities as measured by their long and short diameters (Figs. 4, 5). In this group, 4 cavities showed a decrease in size of from 10 to 25 per cent; they measured originally 6, 7, 11, and 18 cm. Fourteen cavities decreased 25 to 50 per cent in size and originally measured 4, 5, 5, 5, 5, 5, 6, 7, 10, 10, 11, 12, 14, and 16 cm. Six cavities showed a decrease in size of over 50 per cent and originally measured 3, 4, 4, 4, 4, and 9 cm.

Five cavities were distorted in shape by the irregular collapse and the pleural adhesions. One cavity showed no change in size after the collapse but the fluid in it showed a higher level. One cavity could not be seen originally, but was evident after the pneumothorax (Figs. 16, 17); two cavities were accentuated (Fig. 18), and five cavities were obscured by the pneumothorax.

The factors influencing the decrease in the size of a cavity were noted under the headings of adhesions, infiltrations, bronchial status, and the size of the cavities.

(1) Pleural adhesions about the cavity:

Large number and dense adhesions—11 cases—average approximate decrease in size 25 per cent.

Moderate number of adhesions—11 cases—average approximate decrease in size 45 per cent.
Small number of adhesions—2 cases—average approximate decrease in size 70 per cent.

Thus the fewer the adhesions the greater was the reduction in the size of a cavity.

(2) Infiltration about the cavity:

Moderate infiltration—6 cases—average reduction 40 per cent.
Dense infiltration—18 cases—average reduction 40 per cent.
Slight infiltration—no cases.

Thus, the density of infiltration was no factor. A larger series may, of course, have given different results.

(3) Status of the bronchi:

Five cavities were necropsied. Three cases with normal bronchi followed down to about a 1 mm. diameter showed 40, 35, and 60 per cent cavitary collapse; two cases having a tuberculous bronchitis showed 15 to 25 per cent collapse.

Eighteen cases containing 19 cavities were not necropsied but had post-mortem bronchograms. Fifteen cavities surrounded by normal bronchi showed an average collapse of 35 per cent and

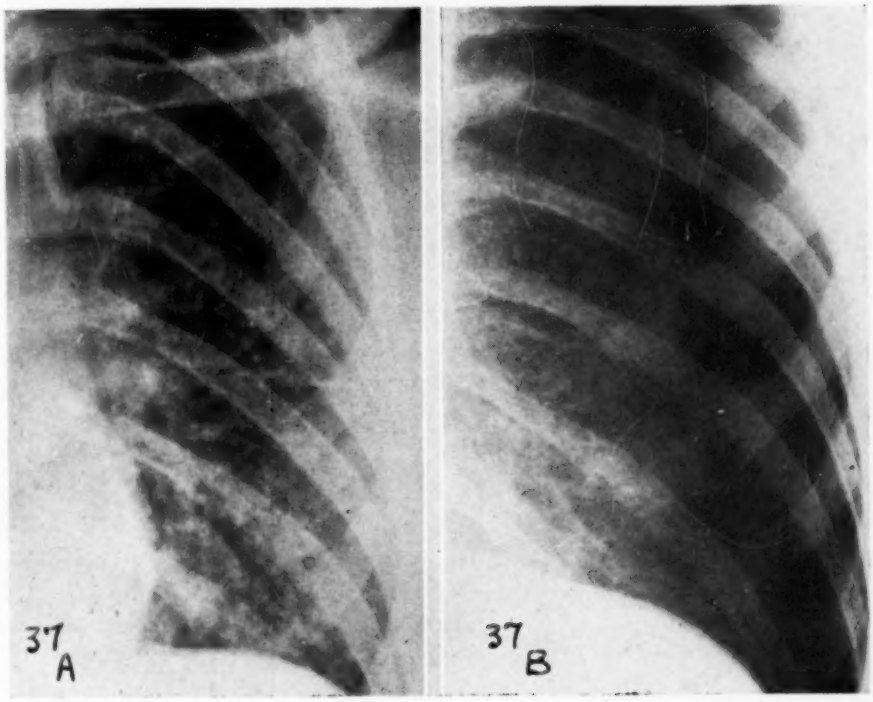


FIGURE 15, CASE 37: Section A shows the postmortem film with a coarse nodular lesion at the right base. Section B shows a pneumothorax with obscuration of the lesion and good aeration of the collapsed lung. Autopsy confirmed the presence of the lesion.

three cavities with abnormal bronchi showed an average collapse of 20 per cent.

(4) Size of the cavities (long diameter):

Up to 4 cm.—6 cavities—average decrease in size 65 per cent.
From 4 to 8 cm.—9 cavities—average decrease in size 25 per cent.
Over 8 cm.—9 cavities—average decrease in size 30 per cent.

Again—Cavities showing a reduction in size:

Up to 25 per cent—4 cavities—Original average size 10 cm.
From 25 to 50 per cent—14 cavities—Original average size 8 cm.
Over 50 per cent—6 cavities—Original average size 4 cm.

Thus, the smaller the cavity the greater was its probable reduction in size.

In summary, it appears that the smallest cavity reductions occurred in the presence of many adhesions, diseased bronchi, and large cavity sizes. Conversely, the best reductions were obtained when there were few adhesions, when the bronchi were normal, and when the cavity was small. We feel that we might have "closed" more cavities if we had encountered more cases with less adhesions.

### *Changes in Pulmonary Infiltrations*

These changes were noted under the headings of obscuration, accentuation, and concentration of the infiltrations after pneumothorax.

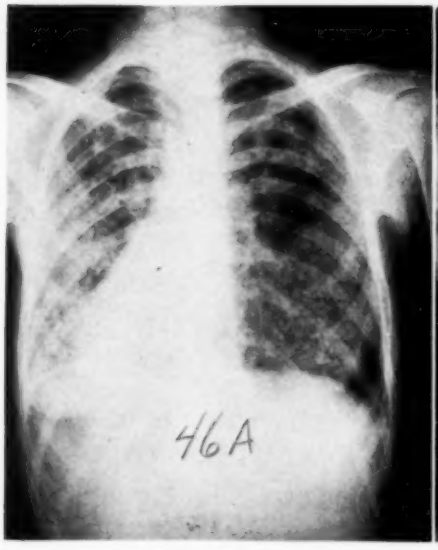


FIGURE 16

FIGURE 17

*Figure 16, Case 46:* Postmortem film.—*Figure 17, Case 46:* Bilateral pneumothorax. The right lung shows selective collapse and concentration of the infiltrations; a bronchogram shows good and normal filling of bronchi and alveoli. The left lung shows a cavity with a fluid level not seen previously. Note the shape and position of the heart.

*Obscuration:*

In 10 cases mild, moderate, and even large infiltrations were much less evident after the pneumothorax (Fig. 15). In 4 more cases a light, scattered infiltration could not be seen at all after the collapse. The existence of these infiltrations was proved in the 6 cases which came to necropsy.

The possible factors were:

(1) In some cases there was actually an increase in the roentgenological surface area of the lung after a partial pneumothorax. The diaphragm, in its descent, pulled the base of the lung downward and produced a better apparent aeration of the lung and a greater pulmonary volume.
(2) The pleural air may have had an obscuring effect on some infiltrations.
(3) One case showed a considerable degree of emphysema.
(4) One case showed an increase in the lung area with only a small pneumothorax and little diaphragmatic motion but with a considerable displacement of the heart pulling the lung with it.

In all these cases one or more diameters of the lung were increased in size or, one may say, the lung was actually stretched

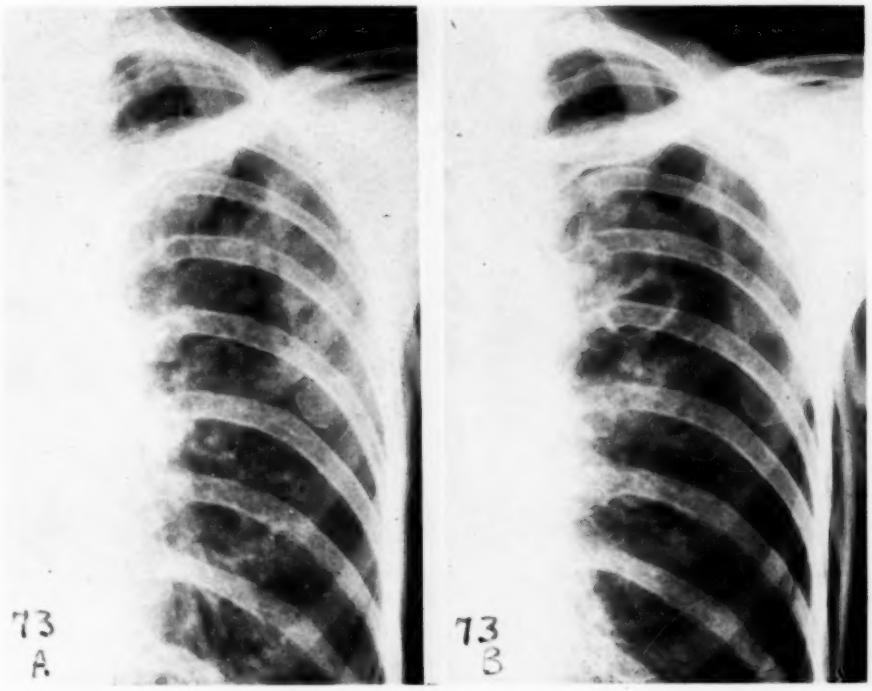


FIGURE 18, CASE 73: Section A shows the postmortem film. No cavity could be definitely seen. Section B shows a right pneumothorax and a definite cavity.

out during the collapse and the apparent increase in aeration may have caused an "emphysema effect" on some of the infiltrations.

*Accentuation:*

In 9 cases the infiltrations could be seen more easily after pneumothorax. Both discrete nodular groups and large areas showed this phenomenon.

The possible factors were:

(1) Several cases showed a greater collapse of the more normal lung and the lesser collapsed areas of disease were more prominent.

(2) In some, the more normal lung became more radiolucent after the collapse due to the stretching of the lung by the diaphragm and the nodules appeared more distinct due to contrast with the surrounding aerated tissue.

(3) No apparent cause.

*Concentration:*

This was noted in 22 cases (Figs. 16, 17). The causes were:

(1) Approximation of the diseased areas by the collapse of the more normal lung tissue between them.

(2) Selective collapse of diseased areas and fusion of shadows.

(3) Collapse of the lung generally to occupy a smaller space.

### *Bronchial Changes*

The trachea was often displaced to the opposite side by the pneumothorax. The bronchi were approximated and compressed. In no case was kinking or distortion produced in the larger bronchi. In some cases, when a bronchogram was made prior to the pneumothorax, the very fine bronchi were completely compressed and emptied of the solution, the larger bronchi were narrowed, and the opaque fluid was pushed up into the trachea.

The bronchi were outlined in 155 lungs and in 44 (or 28 per cent) bronchiectasis was present in the bronchi distal to the main cavernous portion of the disease.

### *Effect of the Pneumothorax on the Contralateral Lung*

Of the 39 cases with bilateral pneumothorax, 8 cases showed a decrease of the first pneumothorax when the second pneumothorax was begun. This was due to the shift back of the heart and lung to a more normal position (Figs. 7, 8, 12, 13, 14).

Three cases with uncollapsed lungs showed a higher fluid level in their cavities after contralateral pneumothorax (Figs. 9, 10). In both cases there was a marked displacement of the heart which may have been the cause.

Three cases with pneumothorax and a marked mediastinal shift showed accentuation of the infiltrations in the non-collapsed lung.

*Pleural Cavity Changes*

In six cases, prior to collapse, the costophrenic angles were clear and no pleural fluid evident. However, after the pneumothorax, fluid was noted and was estimated in the six cases to be from 50 cc. to 100 cc. (Figs. 4, 5). The diaphragmatic pleural reflection showed a normal appearance.

*Pneumothorax Pressures*

During the pneumothorax the pressure used was high and often reached 100 to 200 cm. $H_2O$. The end-point of pulmonary collapse was usually indicated by a leak back of air into the subcutaneous tissues. After the pneumothorax was completed the final pleural pressure was not high. In 8 cases with a uniform collapse the final pressure averaged 9 cm. $H_2O$ (0, 4, 6, 8, 9, 10, 12, and 21 cm.). In 15 cases with irregular collapse the final pressure averaged 10 cm. $H_2O$ (0, 0, 0, 0, 3, 4, 5, 6, 8, 9, 12, 16, 24, 24 and 34 cm.).

*Aspiration of Pulmonary Cavities*

In over 30 cases a needle was inserted into the cavity through the chest wall and attached to a 30 cc. or 50 cc. syringe either directly or by an intervening rubber tube. The cavities varied from 4 cm. to 12 cm. in diameter. Over 20 of these cavities decreased in size when air was sucked out rapidly with the syringe. This decrease in size amounted to 50 per cent or more and lasted from 5 to 60 seconds, after which the cavity returned to its original size. The cavity wall, in many cases, became perceptibly thinned during this decrease in size and the surrounding tissues filling the space became highly translucent.

The factors involved in the phenomenon were:

(1) *Size of the draining bronchus:* In most of these cavities the bronchus was outlined by an intracavitary injection of a barium mixture which would always overflow into the bronchial tree. This would show the size of the bronchus and also the air bubbling from the trachea into the bronchus and then into the cavity shortly after suction was made. The smaller the bronchus the longer was the period of time the cavity remained small. In a number of cases the bronchus was partially obstructed with a very thick barium mixture and, in these cases, the cavity would remain small in size up to five minutes—until enough air was sucked into the cavity through the bronchus to allow it to regain its original size.

(2) *The rapidity and degree of suction:* The more rapidly the air was sucked out the smaller the cavity became—again, until enough air came in via the bronchus.

(3) *State of the nearby tissues:* By this term we include both the cavity wall and the surrounding lung. That the cavity wall was not all caseous tissue, but in many cases was composed chiefly of collapsed alveoli, was shown by the thinning of the cavity wall during the decrease in the size of the cavity. This occurred in even very large cavities. The degree of decrease in size was influenced also by the amount of normal lung tissue about the cavity. Normal tissue was necessary to fill up the space left void by the shrinking of the cavity.

Thus, the narrower the bronchus, the greater the suction, and the more normal adjacent lung tissue present, the greater was the decrease in the size of the cavity subjected to aspiration.

### *Discussion*

Just as in pneumothorax in the living, the post-mortem effects on the *bony thorax* included an elevation of ribs, sternum and clavicle, a widening of the intercostal spaces, and an increase in the frontal and lateral diameters of the thorax. In the living, the process is due to neuromuscular action. In the dead thorax, the mere increase of intrapleural pressure caused the upward motion of the ribs since that is the only direction in which they could move. The same phenomenon is seen in dead animals whose lungs are inflated and deflated through an endotracheal cannula; during inflation the ribs are elevated and the other effects follow. Thus, the inspiratory movements of the chest cage may be produced not only by neuromuscular elevation of the ribs but also by the mechanical increase in the intrapleural pressure and by inflating the lungs.

The descent of one leaf of the *diaphragm* during post-mortem pneumothorax affected the other leaf to only a very slight extent. The same picture is seen in pneumothorax in the living and after paralysis of a leaf. After a phrenic nerve interruption, the paralyzed hemidiaphragm has its action determined by pleural and peritoneal pressures and is not directly affected by the other leaf. Since the post-mortem effects are mechanical in nature, it stresses the fact that the diaphragm may be regarded functionally as two separate muscles, each leaf having not only an independent nerve supply coordinated in the central nervous system but also an independent mechanical action due to its peculiar anatomical structure.

The *heart* showed a surprising degree of mobility and pliability

in this study. A pneumothorax displaced the heart to the other side and a contralateral pneumothorax replaced it in its original position. Descent of one leaf of the diaphragm caused little or no descent of the heart, but a descent of both leaves pulled the heart downward and changed its shape. The picture so produced resembled that seen in emphysema, a small "drop" heart even when it was actually enlarged, and again stresses the purely physical aspects involved.

Pleural *herniation* through the anterior and posterior mediastinums behaved post-mortem similarly to that seen in living cases. It occurred frequently and was reduced by a contralateral pneumothorax. Both anterior and posterior mediastinal "weak spots" were crossed by the pleural membranes under increased intrapleural pressure and this mechanical action is probably sufficient to explain its occurrence in the living patient.

As expected, the type of *collapse* showed great variations due largely to the number and extent of pleuro-pulmonary adhesions and, in relatively free pleural spaces, surprisingly good collapses were obtained. In the living person, pneumothorax causes a diminution in the lung volume due to its elasticity and tendency to retract to its solid airless capacity. And in this study, the dead lung was sufficiently elastic and was reduced to a smaller volume by the positive pressure pneumothorax forcing out much of the air from the alveoli. In both the living and dead lungs there was a reduction of alveolar air: in the living, by the retraction of the lung caused by the increase in intrapleural pressure even though at a subatmospheric level; in the dead, by a slight initial subatmospheric retractility followed by hyper-atmospheric pressure forcing out the alveolar air. Thus, the mechanism of pulmonary collapse has similar features in both living and dead.

The *factors* determining the degree of pulmonary collapse included the pleural adhesions mentioned above and several intrapulmonary ones. Thus, in a relatively free pleural space, the degree of pulmonary infiltration and the size of the pulmonary caverns had a direct bearing on the collapse. Lungs with a mild infiltration showed an average collapse of 60 per cent whereas those with extensive disease showed a 45 per cent collapse. Cases with no pulmonary cavities showed an average collapse of 60 per cent and those with cavities showed a lesser collapse depending directly on their size. Conversely, the greater the amount of normal tissue present the greater was the collapse obtained. The inherent elasticity and retractility of the lung was undoubtedly a factor but could not be measured. The time that the pneumothorax was done, which ranged up to three hours after death, was not a factor in this group of cases.

*Selective pneumothorax collapse* occurred in 14 lungs and presented a picture similar to that seen in the living. Two lungs showed a pre-existing lobar atelectasis. In the four cases autopsied the bronchi to the selectively collapsed lobes were grossly normal and patent. Extensive disease was present in 12 of the lobes selectively collapsed, miliary disease in one, and a light infiltration in one. The selectivity of the collapse occurred with even small amounts of air and was strikingly demonstrated fluoroscopically. One infers that either (1) it was easier to force the air out of the involved lobe, or (2) the tissues in that lobe were under greater elastic tension and so retracted first and most. In the 12 cases with extensive disease the remaining normal tissue in the lobe and the few remaining elastic fibers must certainly have been under greater than ordinary tension and so collapsed first and most when released by the pneumothorax. Why the two cases with miliary disease and the light infiltration showed the same phenomenon cannot be explained. It is our impression that the selective collapse seen in this study was due to the increased elastic tension of the normal tissue about the diseased areas and was present prior to the collapse.

There are several theories regarding the mechanism of selective collapse in the living patient and some stress the absorption of alveolar air and the respiratory motion of the lung. Of course, selective collapse as seen in this group of dead lungs may not be identical with that seen in the living for, in this series, selective collapse occurred almost instantly whereas in the living it may be immediate or it may be delayed for several weeks. The purely mechanical explanation of the normal tissue under greater stretch would apply to the group showing immediate selective collapse. It is more difficult to explain the delayed group unless one supposes that elastic tissue changes take place later in the involved lobe due to increasing fibrosis, slowing of the lymph flow, and anoxia of the tissues. At any rate, it stresses the mechanical factor involved in selective collapse as certainly the dominant one if not the complete cause in itself.

The term *atelectasis,* as used here, denotes that a lobe is decreased in size, appears "roentgenologically airless," and is seen as a dense homogeneous shadow. This occurred in 6 cases in this study and, in 2 of them, it existed prior to the pneumothorax. Whether all these cases showed a true complete airlessness anatomically is not known but the one case necropsied did show it. The other 5 cases presented the characteristic roentgen features seen in typical examples in the living. Some cases of lobular atelectasis may well have occurred also but were excluded to avoid confusion. The chief features present were (1) extensive disease

in the lobe involved, (2) the bronchi could not be filled by bronchography in 4 cases, showed ectasis in one case, and were normal and patent in one case, and (3) the intrapleural pressure over the involved lobe was greater subatmospherically in the 5 cases in which measurements were made.

In the living patient, atelectasis is caused by either bronchial obstruction or by occlusion of the arterial blood supply. However, some cases seen in chronic pulmonary disease show no obvious bronchial block or vascular interference and may be due to an old transient bronchial block, or to a collapse of the alveoli resulting from a blocking of numerous microscopic bronchioles, or perhaps other obscure causes. In the cadaver bronchial block should prevent the escape of alveolar air and certainly no air could be absorbed by the blood. One must suppose then that, in these cases, the atelectasis must have existed prior to the pneumothorax and was only accentuated by it. Any air present in the lobe occurred in cavities and in more normal segmental sections. The existence of a higher subatmospheric pressure over the lobes involved shows that they were already under great elastic tension. The pneumothorax released this tension and the atelectasis became more pronounced.

The normal *parenchyma* of the lung showed a similar roentgen appearance under post-mortem pneumothorax to that seen in living cases. Some cases showed a better aeration of the non-collapsed areas after the pneumothorax than previously, due apparently to the stretching of the lung by the diaphragm and its increase in an apex-base direction. The alveolar air was in direct communication with the atmosphere as shown by the ease of filling the alveoli with radiopaque solutions.

*Cavity changes* were common. Only one small cavity was "closed" in this series and 24 were made appreciably smaller. The greater the size of the cavities and the more extensive the pleural adhesions the less was the decrease in size under collapse. The presence of diseased bronchi also limited the decrease in the size of the caverns. The degree of infiltration about the cavities proved to be no factor but we feel that a larger series may have given different results. The rigidity of the cavity walls was undoubtedly an important factor but was not evaluated. Under more favorable conditions more cavities might have been "closed." It stresses the mechanical factors in the reduction of the size of the cavity post-mortem. Its application to the living pathology may be considerable. In the living patient, cavity closure is often a complex process and this study stresses the common concept that, at least, the initial reduction in size with pneumothorax (or even other

collapse measures) is due to the mechanical release of the tension of the tissues about and forming the cavity wall.

As often seen in the living patient, some cavities showed distortion by pleural adhesions, elevation of their fluid levels, or were made more evident, or were obscured. The practical applications of these phenomena in interpreting disease processes by roentgen films in the living patient is evident.

Changes in the *pulmonary infiltrations* under pneumothorax occurred commonly. The *disappearance* of some infiltrations after pneumothorax was due probably to an "emphysema" effect caused by an actually better aeration of portions of the lung and the presence of air in the pleural cavity. The actual existence of these infiltrations was proved at necropsy. The clinical application is obvious in that such an event might be regarded as the rapid healing of a lesion under collapse. The *accentuation* and *concentration* of other lesions was discussed in the text.

The pneumothorax caused a displacement of the trachea similar to that of the heart. The larger *bronchi* were narrowed and approximated, as seen by the instillation of radiopaque solutions. The very small bronchi were often completely shut out by the approximation of their walls. No kinking or distortion occurred in larger and medium-sized bronchi. The main site of the parenchymal disease showed some bronchial abnormalities in almost all cases and the distal and less-diseased areas (usually lower lobes) showed bronchiectasis in 28 per cent of the lungs.

The effects of the pneumothorax upon the *contralateral lung* were slight and negligible in most cases but several cases showed appreciable changes such as accentuation of infiltrations and a higher fluid level in cavities. The marked displacement of the heart in these pneumothoraces may well have been the cause.

The *aspiration of cavities* post-mortem is of interest. A large number of the cavities were reduced in size for short periods of time. The factors involved were discussed in the text. The several important observations in this experiment include (1) the number of even very large cavities that were reduced in size, (2) the greater the bronchial occlusion the smaller the cavity became, (3) many supposedly thick, rigid, caseous cavity walls became markedly thinned and then returned to the original state, and (4) the surrounding normal alveolar tissue enlarged to fill up the space left by the decreasing cavity.

The thinning of the thick caseous walls demonstrated the surprisingly large amount of atelectatic alveoli forming the wall—much more than was suspected even at necropsy in both gross and histologic studies. This factor may have an important bearing in the living—in the production and healing of cavities, in the

rapid changes in the size of cavities, in the changes in thickness of cavity walls, in changes in the intrinsic pressure mechanics in the lung caused by the collapse and aeration of such areas, and in the effects such changes may have upon the physiology of the cavity wall and its vascular relationships. The ability of the surrounding normal tissue to enlarge and present a picture of "localized compensatory emphysema" may add to the probabilities mentioned.

## SUMMARY

1) Artificial pneumothorax was produced in cadavers and many of the results were identical with those seen in pneumothorax in living patients.

2) The chief effects so produced included mobility of the chest cage, descent of the diaphragm, mobility of the heart, transmediastinal herniation, selective collapse, atelectasis, reduction in the size of pulmonary cavities, and changes in parenchymal infiltrations.

3) These findings contribute to the physics of pneumothorax and stress the importance of the purely mechanical factors involved.

4) The clinical application of these observations are of particular value in the roentgen interpretation of disease processes and changes.

## RESUMEN

1) Se produjo neumotórax artificial en cadáveres y muchos de los resultados fueron idénticos con los que se obtienen en el neumotórax en pacientes vivos.

2) Los principales efectos así producidos incluyeron movilidad de la caja torácica, descenso del diafragma, movilidad del corazón, hernia transmediastínica, colapso selectivo, atelectasia, reducción en el tamaño de cavernas pulmonares y alteraciones en infiltraciones del parénquima.

3) Estos hallazgos contribuyen a nuestros conocimientos físicos del neumotórax y recalcan la importancia de los factores puramente mecánicos que los producen.

4) La aplicación clínica de estas observaciones es especialmente valiosa en la interpretación de procesos y alteraciones morbosas en la radiografía.

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# The Pathogenesis of Primary Atypical Pneumonia

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The term atypical pneumonia, etiology unknown, includes the following: Atypical pneumonia with leukopenia, atypical bronchopneumonia of unknown etiology, acute interstitial pneumonia, virus pneumonia, acute pneumonitis, pneumonitis, disseminated focal pneumonia, and acute influenzal pneumonia.

The disease has come to be written about only within the past decade. It gained widespread recognition in World War II. Because of its nature and close similarity to other respiratory diseases, just where and when it first existed is at present unknown. It has been stated that it is probably not a new disease, inasmuch as a description of a closely similar malady dates back seventy-five years.

The etiology of atypical pneumonia is obscure. It is thought by many that a filtrable virus is the etiological agent but this hypothesis has not been proved. Others believe that two agents, one presumably a virus and the other a bacterium may possibly act in concert to produce an infection different from that which would be produced by either alone. Still others feel that several etiological agents are involved and that therefore primary atypical pneumonia is a syndrome rather than a true entity. Most cases reported are in the young adult population, both male and female. The great majority of cases are in young males in the second and third decades of life.

The cyanosis and dyspnea sometimes noted when the pulmonary process is extensive and bilateral appears to be due to *anoxemia* rather than toxemia such as seen in cases of lobar pneumonia. Some patients do not appear ill at all. Often we are surprised at the paucity of clinical and physical findings when correlated with x-ray evidence of considerable consolidation and early there is apt to be a normal pulse and respiration.

The fundamental pulmonic lesion is an acute interstitial pneumonitis. The pathological change in the involved portion of the lung is essentially a serofibrinous exudation with secondary inflammatory hyperplasia. Only an interstitial involvement in the lung would produce such a combination of roentgen and physical

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findings. First, the alveoli would be air bearing early in the course, giving essentially normal pulmonary resonance and no rales, but the interstitial fluid would add to the density of the lung as seen in the roentgenogram. Second, by swelling, and stasis, eventual seepage into the alveoli producing the moist rales and the "wet lung" picture could occur. Third, the interstitial swelling with stasis and the wetness of the alveoli would account almost quantitatively for the anoxemia, cyanosis, and dyspnea whenever they occur. Fourth, that diapedesis does not occur with subsequent consolidation within the alveoli is attested by the presence of muco-tenacious sputum only occasionally tinged with bright red blood instead of the typical rusty sputum seen in pneumococcus pneumonia. Fifth, the absence of toxic action on the pulmonary capillaries' bed and the low systemic toxicity are in the same direction. Sixth, this series of events is consistent with the roentgen findings, throughout the course of the disease. Seventh, in these described pneumonitic areas, mixed bacterial flora from the upper respiratory tract find a tissue of lowered resistance and an excellent culture media for secondary invasion. The pathological process may then center about the bronchioles, which become filled with pus and desquamated cells from the lining, which is partially or completely destroyed. Classical physical findings of consolidation are not found.

There is no definite characteristic roentgen appearance of this disease of the lung. The density is hazy, soft and extends outward from the hilus well into the parenchyna, occasionally reaching the periphery. The appearance is that of a confluent mottled or rounded area, usually of homogenous moderate density in the central portion, with the borders fading into the normal lung. It is similar in roentgen appearance to the exudative inflammation around a lung abscess. The density is not as marked as that seen in cases of lobar pneumonia; nor is the periphery of the lesion as well defined. The opaque areas suggest air bearing lung.

Cold agglutination tests are positive in a great majority of individuals suffering from primary atypical pneumonia. The technique of testing for cold agglutinins varies a great deal with different technicians but in general the test becomes positive in a "diagnostic" dilution between the third and seventh day of the illness. The maximum titer of the agglutinin is reached in two weeks following which a decline in strength is noted.

Therapy, other than that of a supportive nature, has proved discouraging. Convalescence is often delayed. Slow resolution of the pneumonitic areas and migration to previously unaffected areas are common. Treatment which would prevent either, would materially reduce the man hours lost through this disease.

From a consideration of the above fact, it is postulated that the changes in atypical pneumonia can be explained on a mechanical basis rather than on an infectious one, although secondary bacterial contaminations and complications in the involved lung can and do occur.

In 1943, while having the dual experience of being in charge of a blood bank, as well as the observation of patients on a pneumonia ward; I was impressed with the cold agglutination which sometimes interfered with our crossmatching of atypical pneumonia patients needing blood transfusions. These reactions often occurred at room temperatures. On the ward I was also impressed by the fact that dyspneic patients suffering from atypical pneumonia often did poorly in oxygen tents in which the temperature was quite low. The pneumonia, often, under these circumstances spread rapidly and migrated to uninvolved portions of the lung and to the opposite lung. In a study of fatal cases of the disease in the literature one is further impressed by the fact that these patients were placed in oxygen tents where the pneumonitic process increased until death resulted.

It is therefore postulated that the immediate precipitating factor in atypical pneumonia is autohemoagglutination in the patient's own lung capillaries.

Cold agglutinins have been found to have a wide thermal range of reactivity. Of the large internal organs, the lungs hold the unique position of being in intimate contact with the atmospheric air through the respiratory passages. The pulmonary capillaries form the richest capillary network of the body, the meshes of which are smaller than the vessels themselves. These plexuses lie immediately in the walls of the alveoli either directly exposed to the air or lying under the thin lining epithelium of the alveoli. The network of capillaries form a single layer which is usually common to two or more adjacent alveoli. During quiet respiration, the tidal air amounts to about 500 cc., which is about ⅛th of the total amount that can be respired. This fresh atmospheric air reaches the alveoli by diffusion which allows adequate time for the air to be warmed to body temperature. The minute vessels of the lung are thus unlike those of any other vascular area, surrounded by and practically in contact with air. As severity of exercise increases, hyperpnea results and merges into dyspnea. In healthy individuals the total amount of air drawn into the lungs with each inspiration may be equal to the tidal, complementary, and supplementary air or 4,000 cc. This dilutes a residual air of perhaps 1,000 cc. The total minute volume of air may be as much as 160,000 cc. After such deep inspirations, the respiratory bronchioles and alveolar ducts receive fresh atmospheric air. The

advancing margin of the latter however, as a result of its spike form, is prevented from coming in direct contact with the respiratory epithelium by a layer of vitiated (alveolar air). However because rapid diffusion and mechanical mixing the temperature of the air in contact with the pulmonary capillaries must be lower than body temperature. As a corollary of this, the lower the outside atmospheric temperature the lower must be the temperature in contact with the respiratory epithelium at a given minute volume of respired air, if this minute volume is greater than that in a resting state.

The fallacy of assuming that in vitro test results with cold agglutinins are comparable to vivo findings, is at once apparent. Agglutination in a 1 to 8 dilution of blood plasma is considered significant. However, such dilution does not occur in vivo. Another point which is not often considered is that in vitro tests for cold agglutination the red blood cells are often 1 to 40 or 50 the number they are in the body itself. Capillaries are very small, ½ to 1 mm. in length and up to 10 microns in diameter. A capillary may be so contracted that a red blood cell is unable to enter without undergoing deformation. A red blood cell pressed into an elongated or sausage-like form can frequently be seen within a capillary or even imprisoned by the complete closure of the capillary lumen at each end. In other instances constrictions of the vessel excludes the corpuscles entirely from a neighboring capillary channel, the plasma alone being permitted to pass. This phenomenon is called "plasma stripping" and concentrates increased proportions of red blood cells in other areas. The ready distensibility of the vascular bed of the lungs accounts for the fact that little or no rise in pulmonary arterial pressure occurs during muscular exercise, despite the great increase in cardiac output. It is believed that a fair value for pulmonary capillary blood pressure is 10 mm. of mercury and that this pressure in the lung capillaries is extremely hard to disturb because of the immense area of the vascular net and the readiness with which expansion can be induced. Since the red blood cells barely make their way through the capillaries any increased agglutinative tendency or stickiness caused by contact with air cooled below body temperature may cause them to block a capillary channel.

This process would be different from atelectasis in that it is not a collapse and early the alveoli are open. Such a process would begin a vicious cycle and tend to procreate itself.

Thus we see that in dyspnea the pulmonary capillary blood pressure already low, is not raised and the blood continues its slow course through the lungs. The air in contact with these capillaries may be lower than body temperature and the degree

is related to the temperature of the outside atmospheric air and the minute volume of respired air. A cold agglutinogen of the red cells is activated by this lowered temperature and agglutinates in the capillary; the process becomes wide spread and we have primary atypical pneumonia. The process extends outward from the hilum since, as the air passes toward the periphery of the lung, it becomes increasingly warm. If affects young males mainly since they are more apt to have vigorous exercise out of doors than other groups.

Hemorrhagic areas (hemorrhagic encephalitides), have been noted in the brain and other areas in fatal cases of atypical pneumonia. These could theoretically be caused by clumps of red blood cells washed from the lung capillaries and lodging in the various localities.

Perhaps we will find that cold agglutinins are much like the Rh factor. A certain proportion of individuals may have cold agglutinins and agglutinogens, not detected by present day laboratory methods, and these become activated under certain conditions to produce pathological changes in the lung, or perhaps certain bacteria and viruses cause cold agglutinins to appear and indirectly are responsible for the pathological changes. The marked activity of the autohemagglutinins is dependent on an increase in the concentration of some normal serum constituents. This may occur in response to infections with a variety of agents and may be related to the tissues involved.

If this hypothesis is true, and it can fairly easily be proved or disproved, it is an important contribution to the field of medical science. Treatment of those individuals with atypical pneumonia by their breathing air warmed to body temperature would prevent migration of the pneumonia to other uninvolved areas, and slow resolution of involved areas. Prophylactic treatment could prevent many cases from developing.

## SUMMARY

1) A new concept is presented to explain the pathogenesis of primary atypical pneumonia.

2) Supportive evidence for this theory is introduced from various branches of medical science.

3) It is believed that this presented concept can be fairly easily proved or disproved where cases of atypical pneumonia are available for study.

4) If this concept of the development of atypical pneumonia is correct, it will be an important contribution to present day medical science.

5) Methods of treatment and prophylaxis of atypical pneumonia

will proceed on firm ground once a knowledge of the pathogenesis of the disease is obtained.

## RESUMEN

1) Se presenta un nuevo concepto para explicar la patogenia de la neumonía atípica primaria.

2) Se aducen pruebas obtenidas de varias ramas de la ciencia médica que sostienen esta teoría.

3) Se opina que el concepto presentado puede ser fácilmente comprobado o confutado cuando existen casos de neumonía atípica que pueden ser estudiados.

4) Si este concepto del desarrollo de la neumonía atípica es correcto será una contribución importante a la ciencia médica actual.

5) El tratamiento y la profilaxia de la neumonía atípica reposarán sobre una base firme cuando se obtengan conocimientos de la patogenia de la enfermedad.

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# The Significance of Pleural Effusions as Indicating the Presence of Abdominal Disease*

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About five years ago we were impressed by the frequency with which we were seeing individuals with abdominal distress in whom we found inflammatory, malignant, or nonmalignant lesions of the organs of the abdominal cavity, with or without ascites, and associated with the following physical signs at either base of the lungs:

1) Decreased tactile fremitus,
2) Impaired resonance to flatness, not altered by deep inspiration,
3) Increased resistance to the percussing finger in these areas,
4) Seldom was there any marked change in the breath sounds, except at times there was a slight diminution in their intensity,
5) There was no alteration in voice transmission, egophony was not present.

In the early cases that we saw we thought that these were produced by atelectasis due to the accumulation of intra-abdominal fluid elevating the diaphragm. But later on found them in infectious or malignant lesions of the abdominal cavity in which only small amounts or no fluid was present in the abdomen.

For many years, on the medical service at the Buffalo General Hospital, Doctor Nelson G. Russell Sr. was doing many chest taps with vaccum tubes without any untoward results, and finding a high percentage of fluids in the pleural cavity in various conditions. It was his skill in interpreting these physical signs that led us to make these taps with so many positive results. This was being done well before the x-ray became so valuable in revealing the presence of fluid, and was based on the physical signs we have mentioned. It is generally recognized that fluid at the base and early atelectasis are difficult to distinguish from each other, and here the exploratory tap was of greatest value.

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Most textbooks state that it takes between 300 to 400 cc. of fluid to be present in the thoracic cavity to be recognized, and about 600 cc. in the abdominal cavity. We feel rather certain that we have been able to find smaller amounts of fluid present in both cavities, and proving their presence by abdominal or thoracic aspiration. With this background and the group of patients we have mentioned increasing in number, we performed more taps with positive results, and should like to present our methods of examination of the abdomen and chest, technique of collecting the fluid, the character of the fluids withdrawn, and their diagnostic significance.

### *Physical Examination*

In all patients the well-known signs of fluid were sought for, and in many they were present. We found, though, that the following methods were valuable, and we believe have elicited one which may not have hitherto been emphasized. In our experience, it has been easier to demonstrate the presence of a medium amount of fluid in the abdominal cavity by having the patient stand for the performance of the fluid transmission wave. We believe that in the erect position the pelvic cavity is filled above the brim by a quantity of fluid which when the patient is supine fills the abdominal cavity without coming up far enough laterally to produce adequate fluid transmission or shifting dullness. Attention was called to this by Robert L. Pitfield, of Philadelphia, some years ago.

A physical sign that we have elicited in a number of these cases, where a large amount of fluid was present in the abdomen and a smaller or large amount was present in the left chest cavity, is demonstrated as follows: Placing the left hand on the anterior abdominal wall well down towards the pubic region and with direct percussion over the left thoracic cavity posteriorly from the scapula down with the right fingers, when one reaches the level of the fluid in the chest cavity, the percussion impulse is transmitted through the fluid in the chest, through the diaphragm, through the abdominal fluid to the left palpating hand on the anterior abdominal wall. In other words, there is a continuous column of fluid which is an excellent transmitter of percussion waves. This has been of value in demonstrating fluid in both the abdominal and thoracic cavities. We have not elicited this sign as readily when the right chest cavity is involved, due, we believe, to the interposition of the liver.

### *Method of Securing Fluids*

All of our fluids from the chest and abdomen have been secured by using the vacuum tube, or a 20 cc. syringe with a 16

to 18 guage 1½ to 2 inch needle, straight or short bevel. We have withdrawn the peritoneal fluids with the patient in either the standing or sitting position. We have used ethyl chloride spray only for anesthesia purposes in securing fluids. In 104 pleural and 100 peritoneal aspirations we have had no accident due to injury to the lung, pleura or abdominal contents.

We have performed this abdominal tap just before surgery, and have investigated the peritoneal surfaces and the abdominal organs in the vicinity of the tap, and found no evidences of hemorrhage or trauma. Under these circumstances, we believe that it is not necessary to use a trochar to secure fluids for diagnostic purposes from the abdominal cavity. Even when fluid is not present in the abdominal cavity the insertion of a needle of this size and type has not been associated with trauma. In a personal communication from Dr. J. A. Bargen, he stated that following the injection of various vaccines to stimulate the peritoneal response in patients with ulcerative colitis they had not injured the intestinal canal.

In aspirating fluid from the left chest, and in the presence of ascites, one must be sure that the needle is inserted high enough

FIGURE 1

to be well above the level of fluid in the abdominal cavity so as to be certain we are not withdrawing fluid from the abdomen. In none of our cases did we find empyema present.

## SELECTED CASE REPORTS

### *Inflammatory Lesions in the Abdomen Associated with Fluid in the Pleural Cavity and Combined Symptomatology*

The presence of fluid in either pleural cavities is associated with fever, leucocytosis and an increased sedimentation rate when the etiological factor in the abdominal cavity is inflammatory in type. We should like to present the following case of this type to demonstrate these findings.

*Case 1:* A physician, 52 years old, had considerable distress and discomfort of five week's duration, in the left upper abdomen and pain in the left side at the costal margin, urinary frequency, pain in the left lower chest posteriorly not associated with respiration.

Physical examination revealed a temperature of 100°, pulse 83, respirations 22. There was tenderness over the left kidney, impaired resonance at the left base not altered by deep respiration, and a small amount of clear fluid was removed, which on culture and sedimentation studies was negative. White blood count, 12,100; 77 filaments; blood culture, negative; negative urine; negative urogram with skiodan.

X-ray examination of the chest: suggestion of an elevation of the left hemidiaphragm. A flat film of the abdomen revealed some bowing of the spine to the right and a disturbance in the psoas muscle shadow. The diagnosis was made of a perinephritic abscess close to the left diaphragm with fluid formation in the left pleural cavity due to inflammatory reaction beneath the diaphragm. Surgical drainage of the kidney area was done and a perinephritic abscess was found.

This was one of our first cases in which we correlated early the accumulation of fluid in the pleural cavity secondary to an inflammatory lesion beneath the diaphragm. Since this time, we have accumulated 6 cases of fluid in the right pleural cavity due to a subdiaphragmatic abscess secondary to a perforated peptic ulcer, post-cholecystectomy, ruptured appendix, and one due to amebiasis.

### *Right-Sided Pleural Effusion Secondary to a Right-Sided Subdiaphragmatic Abscess*

*Case 2:* A young man of 34, had rheumatic heart disease with aortic insufficiency and duodenal ulcer. Two days previous to his admission to the hospital, he was seized with severe epigastric pain, nausea and emesis, with chills and fever and burning on urination. There was no diarrohea. He had not worked for one year previous, and had never been outside the city of Buffalo.

Physical examination by several examiners revealed tenderness in the mid-epigastrium, more marked over the liver, which was enlarged. He was slightly icteric. There was impaired resonance at the right base. An enlarged liver and spleen developed. The right hemidiaphragm showed

fixation, and there appeared to be a small amount of fluid in the right hemithorax.

The leucocyte count went to 38,000; 60 bands, 36 filaments; icteric index, 20; sedimentation rate, 50; Urinalysis was negative. A pleural tap showed the presence of a clear yellow fluid in the right hemithorax.

Hepatitis was considered as a possible diagnosis. With the appearance of fluid in the chest, which was removed, cultured and imbedded and found to be negative, a diagnosis of an inflammatory lesion beneath the right diaphragm with inflammatory response in the pleural cavity was made. Etiological source being undetermined, a ruptured peptic ulcer was suspected. Drainage of the abscess was instituted by the surgeon, a fistulous tract developed, and while under the observation of the surgeon, its persistence made him suspect that it might be due to amebiasis. He made the necessary studies and demonstrated the presence of motile amoeba in the drainage fluid, and a complete response was made to the administration of emetin.

### *Right-Sided Fluid Due to Perforated Peptic Ulcer*

*Case 3:* A male, 56 years old, with a previous history of a peptic ulcer 25 years ago, and a resection of his caecum for a carcinoma 5 months previously entered the hospital complaining of severe epigastric distress which occurred very suddenly. The abdominal pain was described as being rather steady than sharp, it lasted for 1½ hours when he received a hypodermic injection of morphine. Vomiting of fluid and solid particles, and several loose stools were noted.

The immediate physical examination revealed a small amount of tenderness in the region of the scar of his resection. No diagnosis was made at this time, except consideration was given to acute gastroenteritis, perforated abdominal viscus was considered unlikely. Four days later diffuse epigastric abdominal tenderness was observed, and six days later it was noted that he had considerable hiccoughing. There was limitation of motion of the right hemidiaphragm. Pleural friction sound was heard, and the diagnosis of diaphragmatic pleurisy was suggested. Flat films revealed no free air in the abdominal cavity. Physical examination at that time revealed impaired resonance at the right base with the possibility of fluid considered. Tap performed, and slightly cloudy yellow fluid was withdrawn. X-ray demonstrated partial obliteration of the costo-phrenic angle, some pleural thickening, some streaky atelectasis.

Fifteen days after admission a large subphrenic abscess was drained and subsequently, fluid was found at the right costo-phrenic sinus. The patient returned home in four weeks.

In 8 weeks he was re-admitted to the hospital, and he had definite evidence of dullness and a few rales at the right base. X-ray film showed an obliterative pleuritis as well as a definite fluid level between the liver and the diaphragm. Through the old incision, another small localized abscess was drained.

These three cases are examples of patients originally presenting symptoms of upper abdominal distress and discomfort associated with localized signs in the abdomen and chest, and representing an inflammatory reaction on the pleural surface of the diaphragm

and the pleura covering the lung and an accumulation of fluid in the pleural space. Ochsner called attention in 1,380 cases of subdiaphragmatic abscess, pleurisy with effusion occurred in 388 on the affected side.

### *Abdominal Fluid Associated with Benign Pelvic Tumors*

Woman, 71 years old, with increasing swelling of the abdomen in the past 5 years. Patient noticed a year previously, a sense of pressure and fullness in the lower anterior chest with marked exertional dyspnoea. Had gained 18 lbs. in the past three years; no cough. The abdomen showed definite distension with fluid; liver and spleen not palpable; fluid wave readily demonstrable with shifting dullness. Diagnostic abdominal paracentesis was done, and 50 cc. of clear yellow serum was secured. Examination of the fluid was negative for micro-organisms and malignant cells. Examination of the chest revealed impaired resonance of the left base, some distant coarse rales at both bases, a small amount of fluid was present in the left chest cavity. A firm irregular mass was present in the pelvis. An x-ray film of the chest was negative. The differential diagnosis lay between a malignant tumor of the pelvis with implants on the peritoneal surfaces and pulmonary metastases, or a benign tumor associated with ascites and hydrothorax. On opening the abdomen, 700 cc. of clear serous fluid escaped, and benign bilateral ovarian cystomas were removed.

In 1937 Meigs called attention to a group of cases in which ascites and hydrothorax was associated with fibroma of the ovary. In July 1943, in a subsequent article, he called attention to the fact that in 1937 Muriel B. McIlrath located a report by Lawson Tait, who in 1892 described a patient with left-sided hydrothorax and ascites associated with a large solid growth of the right ovary. The removal of this tumor resulted in a complete recovery. Tait states, "The striking result obtained in this case has gone a long way to confirm me in the advisibility of extending the principles of exploratory incisions in the abdominal disease to an almost universal application."

Since Meigs called attention to this syndrome, a number of these cases have been reported, and the emphasis has been placed upon the fact that the presence of a pelvic tumor and ascites and hydrothorax does not necessarily mean carcinoma in the abdomen with pulmonary metastases. Meigs stated that benign tumors of the ovary should be considered an etiological factor, and may cause a simultaneous collection of fluid in the abdominal and chest cavities.

Our patient represents a combined symptom complex of abdominal distress and thoracic distress with no evidence of vascular or pulmonary disease to account for it, with a small amount of effusion in the left chest and ascites, secondary to a benign tumor of the ovary.

In the complete discussion of the presence of fluid in the abdomen with dyspnoea, abdominal pain, and abdominal tumor, Meigs and others state that it is not known why fluid accumulates in the abdomen and pleural spaces. There is no inflammatory reaction in the abdomen or chest in these patients. Serum protein determinations in his cases and our own have been found normal to rule this out as an etiological factor, and there have been no cardiac or renal conditions present.

Pinner and Moerke, in a careful review of the etiology of pleural effusions, came to the conclusion that the pleura is highly permeable in either direction for the constituents of the blood, but were unable to offer from these findings why such an exchange occurred. Meigs demonstrated by the injection of carbon into the abdominal cavity containing ascitic fluid its rapid passage into the pleural cavity. He believes that the transmission is through lymphatic drainage.

In six cases of cirrhosis of the liver, and in five cases of ascites due to malignant implants on the peritoneal surfaces, fluid was present in the left hemithorax.

In many of the fluids in the abdomen due to a cardiac condition, fluid was present in the left hemithorax as well as in the right. Recently, McPeak and Levine have called attention to the ratio of this finding. Depending on the method considered, right hydrothorax predominated in from 56 to 89 per cent of cases and left hydrothorax in from 12 to 17 per cent. Fluid was equally distributed in 3 to 27 per cent.

### *Analysis of Cases with Fluid in the Pleural Cavity*

By the performance of these taps we were impressed by the number of times we were able to demonstrate fluid in the left hemithorax with a minimal presence of physical signs. We found

TABLE 1

ETIOLOGY OF PLEURAL FLUIDS OTHER THAN TUMOR

| | |
|---|---|
| Arteriosclerotic Heart Disease | 15 |
| Tuberculosis | 6 |
| Rheumatic Heart Disease | 6 |
| Cirrhosis | 3 |
| Pneumonia | 2 |
| Post Operative Embolus | 2 |
| Nephritis | 2 |

*One Each:* Lupus Erythematosus, Ovarian Cyst, Post-Operative Effusion, Hemolytic Anemia, Over-Hydration, Chronic Lung Disease.

the x-ray findings of limited value. Sante called attention to the difficulty in the interpretation of basal exudates of subphrenic origin by x-ray film.

In this group of patients, we had 9 cases of cirrhosis of the liver, 3 of which were proven by postmortem, 6 proven clinically, with a maximum amount of their symptoms and signs abdominal in nature, but in three of whom pleural fluid was present in both the left pleural cavity and abdominal cavity, and demonstrated the fluid transmission sign. An analysis of these fluids for their cellular contents failed to reveal the presence of any cells that could be considered malignant. In 21 cases we were able to secure positive pleural cellular findings, proven by surgery and biopsy in 13, and by the clinical course in 8.

TABLE 2

PLEURAL SEROSAL IMPLANTS WITH EFFUSION

| SOURCE | |
|---|---|
| Breast | 2 |
| Gallbladder | 1 |
| Lung | 1 |
| Leucosarcoma | 1 |

In one of these, the case of leucosarcoma, findings in the effusion made the diagnosis unaided.

In breaking down our figures to show the types of tumor, the following is the result:

TABLE 3

TYPES OF TUMORS

| | No. of Cases | Tumor Cells Found |
|---|---|---|
| Primary Carcinoma of Lung | 22 | 9 (40%) |
| Carcinoma of the Breast | 16 | 4 (25%) |
| Lymphosarcoma of Lung | 8 | 4 (50%) |

## SUMMARY

1) In all cases of pleurisy with effusion the etiological factor of an abdominal lesion must be considered.

2) Small amounts of fluid may be present in either pleural cavity, and may be demonstrable only by physical signs and exploratory aspiration.

3) In a group of abdominal and thoracic taps, using a size 16 to 18 gauge 1½ to 2 inch needle, straight or short bevel, we have had no untoward reactions, either local or general.

4) Five to ten cc. of fluid are adequate to demonstrate the presence of pathological cells.

5) The importance of the presence of fluid in the thoracic cavity with a nonmalignant mass in the abdomen must be emphasized.

6) The x-ray may show only a haziness at either base which is difficult to distinguish between a small amount of fluid, atelectasis, or pulmonary congestion, and aspiration may be the only method of proving the presence of effusion.

7) It is exceedingly rare for an inflammatory lesion in the chest to pass downwards through the diaphragm and involve the abdominal cavity.

8) We have presented a case of a Meigs' syndrome.

9) The increasing number of returned veterans with amebiasis involving the liver makes it necessary for us to remember it as an etiological factor of pleural effusion.

10) The gastroenterologist must carefully examine the thoracic cavity in many diseases involving the abdominal organs.

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We are grateful to Dr. Dorothy Nagel Shaver, Dr. Charles F. Becker and Dr. Kornel Terplan for permission to use their pathological findings and to Dr. Joseph E. Macmanus for report on Amoebiasis case.

## RESUMEN

1) En todo caso de pleuresía con derrame se debe considerar el factor etiológico de una lesión abdominal.

2) Pueden ocurrir pequeñas cantidades de líquido en cualquiera cavidad pleural y es posible demostrar su presencia solamente mediante los signos físicos y la punción exploratoria.

3) En un grupo de paracentesis abdominales y torácicas en las que se emplearon agujas de calibre 16 a 18, de 1½ a 2 pulgadas de largo, no se observaron reacciones contraproducentes, ni locales ni sistémicas.

4) De cinco a diez cc. de líquido son suficientes para demostrar la presencia de células patológicas.

5) Se debe recalcar la importancia de la presencia de derrame en la cavidad torácica con un tumor abdominal no maligno.

6) Es posible que la radiografía solamente muestre una leve sombra en una de las dos bases, la que es difícil de distinguir entre un derrame pequeño, atelectasia o congestión pulmonar, y la paracentesis puede ser el único método de comprobar la presencia de derrame.

7) Es sumamente raro que una lesión torácica inflamatoria pase a través del diafragma e invada la cavidad abdominal.

8) Hemos presentado un caso de síndrome de Meig.

9) El número creciente de veteranos de la guerra que han re-

gresado con amibiasis que afecta al hígado nos obliga a recordar que este es un factor etiológico de derrames pleurales.

10) El gastroenterólogo debe examinar cuidadosamente la cavidad torácica en muchas enfermedades que afectan los órganos abdominales.

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# Tuberculous Infection in the Children of Lima, Peru*

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The high figures of tuberculous infection found in Perú during the last 10 years, in which tuberculosis control in the various communities has been carried out, almost systematically, has shown that our country is passing through a stage of mass infection of tuberculosis.

The investigation, with the purpose of determining the percentage of infection, as well as tuberculosis morbidity, have given figures of infection that fluctuate between 30 and 60 per cent in the first six months of life and 70 to 80 per cent in the adults.

By means of the intra-dermal reaction of Mantoux with 1/1000 and 1/100 old tuberculin of Koch, in more than 100,000 individuals in the Capital of Perú from 1936 to 1946, the following results have been obtained:

Out of 50,000 children from 0 to 6 months of age, 25 per cent of infection was obtained in the first month, and 66 per cent in the sixth month; of 10,000 children from 7 to 12 months, 50 per cent reaction to tuberculin was obtained; of 20,000 children from 1 to 5 years, 60 per cent was obtained; of 10,000 children from 6 to 15 years, 65 per cent was obtained; and of 15,000 children over 15 years, 70 per cent reaction to tuberculin was obtained.

We have tried to find the cause for the high percentage of tuberculous infection during the first six months of life among the children of Lima. We have reached the conclusion that it is due to the lack of systematic examination of the pregnant women, to the lack of special services in the hospitals in order to separate tuberculous patients, to the lack of special services for the separation of the newborn of infected mothers, and to the need of establishing a methodical form of vaccination of the newborn with B.C.G.

Bearing these facts in mind, we have procured the above mentioned services. Through our suggestions, a special service for pregnants in our Maternity Hospital of Lima has been established. We are having systematic control of pregnant women by means of x-ray films. Finally, due to our initiative too, the first hospital for the newborn of infected mothers was just inaugurated. This

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hospital is located in the town of Chosica, 40 Km. from Lima, 900 m. above sea level. All the children of infected mothers, that are born in the Maternity Hospital, after being vaccinated by B.C.G. are sent to this hospital in Chosica.

This work was carried out with the cooperation of the Ministry of Public Health, the Head of the National Antituberculosis Campaign, and the Rotary Club of Lima. We believe this is going to be useful in order to cut down, in a short time, the percentage of infection found in the infants of Lima.

The prevalent concept in my country is that the antituberculosis campaign must begin with the periodical x-ray examination of the pregnants, must follow immediately after birth with the separation of the infected mother, and B.C.G. vaccination. Thus, my country expects to reduce the high levels of infection, morbidity and mortality caused by tuberculosis in infants.

Before finishing, I would like to mention the need for establishing a standard procedure in the kind of tuberculin to be used in testing and determination of rates of infection in all countries, starting with the Panamerican nations. Also, a standard must be set upon the dilutions of tuberculin that is to be used, so it will be possible to make comparison of the results and investigations in every place.

On my part, I may suggest the use of the old tuberculin of Koch. Results obtained in a work were presented at the Tenth Latin American Congress of Tuberculosis, held at Buenos Aires in 1940. We made a comparative study of the degree of intensity to the tuberculin reaction, injecting old tuberculin of Koch in the dermis of the forearm, and P.P.D. in the other forearm of the same person, of the same dilutions. We proved better results, more positive reactions in tests with old tuberculin. These tests were done in more than 2,000 school children between 6 and 15 years of age.

It is equally important to set a standard pattern of the dilutions to be used. We consider that it would be enough to use dilutions 1/1000 and 1/100 by the technique of Mantoux. To the negative cases with 1/1000, a new injection is given with 1/100. Those who are negative to this dilution could be considered as free of infection.

## SUMMARY

The author has done more than 100,000 tuberculin tests in order to ascertain the incidence of tuberculous infection, at a different age of life. He has found 66 per cent infection at the sixth month of life, 60 per cent at the fifth year, and from 70 to 80 per cent in the adult.

The reason for the high percentage found in the first six months of life lies in the lack of special services for infected pregnant women and in the lack of systematic x-ray examination of all pregnants. The institution of such methods and services, the establishment of hospitals for the separation of the new born, previously B.C.G. vaccinated, of the infected mother, are going to aid in the lowering of the high levels of infection, morbidity and mortality, due to tuberculosis, in infancy.

Standardization of tuberculin to be used in the determination of the degrees of tuberculosis infection as well as conventional dilutions are advocated.


## RESUMEN

El autor ha llevado a cabo más de 100,000 pruebas a la tuberculina a fin de verificar la frecuencia de la infección tuberculosa en diferentes edades de la vida. Ha encontrado un 66 por ciento de infección en el sexto mes de vida, 60 por ciento en el quinto año y de 70 a 80 por ciento en el adulto.

La razón del alto porcentaje encontrado en los primeros seis meses de vida se halla en la falta de servicios especiales para mujeres preñadas infectadas y la falta de exámenes radiográficos sistemáticos de todas las mujeres embarazadas. La institución de esos métodos y servicios y el establecimiento de hospitales para la separación del recién nacido, previamente vacunado con B.C.G., de la madre infectada, van a ayudar a bajar los elevados niveles de la infección, morbicidad y mortalidad por tuberculosis en la infancia.

Se aboga por la normalización de la tuberculina que se va a usar en la determinación de los grados de infección tuberculosa, y por el empleo de diluciones convencionales.

# Pneumoperitoneum and Phreniclasia in the Treatment of Pulmonary Tuberculosis

## Therapeutic Observations in 550 White and Negro Cases*

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### *Introduction*

During World War II many discharged soldiers and sailors were received in the United States Veterans Hospital at Oteen, North Carolina, with pulmonary tuberculosis. A large number of these patients were in the younger age group, and unfortunately had extensive bilateral disease, many with cavitation. In the past, the only treatment which could be offered a patient with this amount of disease was bed rest. This presented a serious problem.

It was decided in 1943 to treat this type of case with a phreniclasia on the side most involved, and to follow this with pneumoperitoneum. If both lungs were equally involved, pneumoperitoneum was instituted and the phreniclasia added later on the side with the least lift of the diaphragm. Results were so encouraging, that the treatment was gradually applied in more favorable cases. This article reviews 550 cases treated and endeavors to assay the value of this method of treatment.

In evaluating the statistical data, it is important to bear in mind the fact that the majority of patients in this series had far advanced pulmonary tuberculosis, many with a poor prognosis. It is realized that the time element is very important in evaluating any form of pulmonary collapse, and we therefore feel justified in dividing the cases into two groups, one group having the treatment six months or more, and the other six months or less.

### *Physiology*

Normally, the general pressure within the abdomen tends to equal that of the atmosphere. The subdiaphragmatic region, however, differs from other parts of the abdominal cavity in that the intrapleural negative pressure is transmitted to this area. As

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this subatmospheric pressure in the upper abdomen is gradually changed to a positive pressure by the intraperitoneally injected air, there is an upward pulling force exerted by the intrapleural negative pressure and the diaphragm rises. The elevation of the diaphragm is also influenced by the lifting effect of the air injected.

With the rise of the diaphragm the thoracic capacity is reduced, and the intrapleural negative pressure becomes less negative. This latter change permits a greater contraction of the elastic structures of the lung, and pulmonary relaxation is attained. We see, therefore, that when a phreniclasia and pneumoperitoneum have been instituted, two things are accomplished: first, the high rise of the diaphragm reduces the lung volume and facilitates drainage; second, there is marked relaxation of the lung and bronchial tree. There is little shortening of the bronchi and little narrowing of their lumina during expiration, and this allows for continuous drainage through patent bronchi. Improved drainage and relaxation of the lung are the two outstanding factors which contribute to the excellent results secured in healing the lung with pneumoperitoneum.

Every patient in this series has been asked the question, "What did you notice after the treatment was started?" A common reply was, "My cough and sputum increased in the beginning but it was easier to raise the sputum." Favorable symptomatic response was noted early in nearly all cases. Expectoration was easier and the cough and sputum gradually disappeared. Improvement in pulmonary drainage was followed by a decrease in toxicity and a gradual disappearance of constitutional symptoms; the temperature and pulse returned to normal, the patient began to sleep at night, his appetite improved, and he started to gain in weight.

### *Indications*

1) Phreniclasia and pneumoperitoneum are indicated when the tuberculous lesion is too extensive for bilateral pneumothorax. Patients with extensive bilateral disease, for whom nothing can be offered in the form of surgical procedures, not only secure considerable relief, but in many instances attain a clinical cure.

2) Cases with unsatisfactory collapse of the lung with pneumothorax due to adhesions where a pneumonolysis has been unsuccessful. In these cases the lung has been reexpanded and a phreniclasia and pneumoperitoneum added with favorable results.

3) Pulmonary hemorrhage which can not be controlled by artificial pneumothorax.

4) As a measure to prepare patients for thoracic surgery, who are at the time too ill for radical intervention. Many cases with

scattered pulmonary infiltrations below the cavity area can be cleared up completely, and the apical cavities so reduced in size that thoracoplasty becomes feasible and its extent reduced.

5) Pneumoperitoneum is indicated as an adjunct to thoracoplasty when lesions in the lower portions of the lung remain active, or a new focus develops in the contralateral side.

6) Pneumoperitoneum alone without phreniclasia is the treatment of choice in cases of tuberculous pneumonia. Spontaneous collapse and the danger of empyema make pneumothorax a dangerous procedure.

7) To help obliterate the pleural space in pneumothorax cases when, following discontinuance of the treatment, the treated lung fails to reexpand completely.

8) The treatment of pulmonary tuberculosis in the diabetic patient. The predominantly exudative and caseous lesions which exist in these cases and the frequency of spontaneous collapse during pneumothorax treatment make pneumoperitoneum the treatment of choice.

9) In severe intractable cough. Severe cough which produces physical exhaustion, loss of appetite, vomiting and disturbed rest is relieved. Pneumoperitoneum in these cases is a palliative and justified therapeutic measure.

10) Mid zone cavities, which do not respond to pneumothorax, frequently close with pneumoperitoneum.

### *Complications*

Complications in this series have been surprisingly few and there have been no accidental deaths. The most serious complication is air embolism, but we have not experienced it in any of our cases. Another complication is the passage of air through the aortic or esophageal hiatus up through the mediastinum into the fascial planes of the neck. This occurred in one case. Subsequent treatment was to reduce the amount of air refill and lower the intraabdominal pressure. Treatment has been continued and the complication has not recurred.

Three patients in this series developed appendicitis and were operated upon successfully. There was no evidence of chronic peritonitis attributable to the treatment. The incidence of acute appendicitis in patients treated with pneumoperitoneum does not appear to be any higher than in the rest of the sanatorium cases. Two patients developed peritoneal effusions to a degree requiring aspiration, and treatment with pneumoperitoneum was discontinued. Small asymptomatic effusions were noticed in five cases but these did not interfere with continuance of the treatment.

They were seen to occupy the space between the liver and the lateral abdominal wall when roentgenologic examination was made with the patient in the upright position. Occasionally an effusion may be seen in the space between the stomach and the lateral abdominal wall. Care must be exercised not to mistake this for a fluid level in the stomach.

Peritoneal adhesions were seen in a number of cases but in the majority caused no trouble. In some instances, however, they will prevent a satisfactory rise of the diaphragm, and in six of our cases pneumoperitoneum had to be discontinued as unsatisfactory.

Subcutaneous emphysema during pneumoperitoneum treatment has occurred but is a complication of minor importance. Its chief symptoms are localized pain and palpable crepitation. The resulting discomfort is slight and disappears in a short time. The cause is generally a faulty position of the needle and the injection of air into the abdominal wall instead of the peritoneal cavity. Improved technique by the operator should eliminate this complication entirely.

After the induction of pneumoperitoneum, the patient may complain of pain in the hypochondriac or epigastric area and a referred pain along the phrenic nerve into the neck and outward to the shoulder. If this occurs, we have found it not severe, and it generally does not recur after the first treatment.

Penetration of the intestine or abdominal organs has not occurred to our knowledge. This complication should never arise if ordinary care is practiced.

### *Technique*

The procedure of initiating pneumoperitoneum and giving refills is not difficult and is believed to be much less dangerous than pneumothorax. Following the phreniclasia, we wait three weeks to allow the patient to become adjusted to his temporary phrenic interruption. In selecting the point of entry the proximity of surgical scars should be avoided because of the possibility of intestinal loops being adherent to the abdominal wall. We have selected a routine site to the left of the umbilicus through the rectus muscle. It is felt that the rectus forms a muscular valve and prevents subcutaneous emphysema, especially in patients with considerable cough.

The procedure is carried out with the patient lying flat on his back. The skin is sterilized with tincture of merthiolate, and with strictly aseptic technique the skin and various layers of the abdominal wall are infiltrated with one per cent plain novocain, using a 25 gauge, 1½ inch needle. After adequate anesthesia is secured, a 19 gauge, 3 inch needle is inserted. In the beginning

of the treatment it is advisable to attach a syringe filled with novocain to the needle, and a free flow of novocain from the syringe is a good indication that the needle has reached the free peritoneal space. By using novocain one can avoid the possibility of air embolism. With reference to the latter, it is advisable to draw back on the plunger of the syringe before injecting any air to make sure that the point of the needle is not in a blood vessel. The air is then introduced by means of a Zavod pneumothorax apparatus, which we have found to be the most satisfactory for this type of treatment.

The initial treatment is 450 cc. of air repeated twice weekly for two weeks. This is a trial period to determine how the patient adjusts to the treatment; if it is satisfactory, he is placed on a weekly schedule, and the amount of air refill is gradually increased. The maximum amount of air given at a refill to the average patient, after the pneumoperitoneum is well established, is 900 cc. of air. We endeavor to build up the pressure in the abdomen to a positive pressure of 10 cm. of water and secure a lift of the diaphragm to the anterior third rib. Cases in which this can be accomplished secure the best results. Some patients can not tolerate this pressure, and the best policy to follow is: the amount of air injected should be gauged on the basis of individual requirements and subjective response of the patient during treatment.

There are certain symptoms and signs which can be used as indicators of a successful induction of pneumoperitoneum; these are: referred pain along the phrenic nerve into the neck and outward to the shoulder, and a feeling of tightness in the hypochondriac or epigastric area. The objective signs are: disappearance of liver dullness and the presence of a tympanitic percussion note. Under fluoroscopy one will note the beginning lift of the diaphragm by the air beneath.

Following the initial treatment, the patient is requested to lie flat on his back until bed time. In a few cases the pain was severe and was relieved by elevating the foot of the bed. The patient is also advised to turn over in bed slowly or he will notice a sudden shifting or rolling of the air and an uncomfortable feeling will result; this disappears as the intraabdominal pressure is increased and the pneumoperitoneum becomes well established.

One of the most frequent causes of failure to induce a pneumoperitoneum is failure to insert the needle in far enough. If the point of the needle does not reach the abdominal cavity but is lying in the tissues, the flow of air is very slow and the manometer will show a non-oscillating positive pressure of +15 to +25 cm. of water, and the patient will generally complain of pain at the site of injection.

*Report of Cases*

New cases on admission to the hospital are reviewed by a Therapy Board, after a thorough study and examination on the Reception Service. This Board is composed of the Clinical Director, Chiefs of the Surgical, Medical and X-ray Services and the Ward Surgeon. This plan gives the patient the benefit of the knowledge of a group well trained in the treatment of tuberculosis, and eliminates the error of individual judgment. All cases are routinely reviewed by the above Board every three months, with new x-ray films and sputum examinations, or sooner if conditions arise requiring the Board's opinion.

In this review the Therapy Board's opinion of the status of the case forms the basis of this study. Table I lists what has occurred in the 550 cases treated; there are 410 cases still under treatment, 45 prepared for major surgery, 50 discontinued as unsatisfactory and 45 dead.

The 410 cases still under treatment have been divided into white and negro groups treated six months or more and six months or less. Table II shows 134 white cases with far advanced pulmonary tuberculosis treated six months or more. The sputum has been converted in 106 or 79 per cent. Cavity closure occurred in 80 of 111 cases with cavitation, or 72 per cent. The clinical status of this group finds: 14 arrested, 2 apparently arrested, 35 quiescent, 70 improved, and 13 unimproved. In the group listed as improved there has been clearing of the pulmonary lesions, cavities reduced in size or closed, and toxicity relieved. In the second section of Table II we find 18 moderately advanced cases. The sputum has been converted in 16 or 89 per cent. Cavities closed in 10 of 11 cases with cavitation, or 91 per cent. The clinical status reveals: 4 arrested, 4 quiescent, 8 improved, and 2 unimproved.

Table III reviews the results attained in 76 far advanced and 6 moderately advanced negro patients treated 6 months or more. In the far advanced group of 76 the sputum was converted in 56 or 73 per cent, cavity closure occurred in 37 of 55 cases with cavitation, or 67 per cent. The clinical status finds: 3 arrested, one apparently arrested, 33 quiescent, 29 improved, and 10 unimproved. In the six moderately advanced cases sputum conversion and

TABLE I

Classification of 550 Cases Treated with Phreniclasia and Pneumoperitoneum

| No. of Cases | Still Under Treatment | Prepared for Major Surgery | Discontinued as Unsatisfactory | Expired |
|---|---|---|---|---|
| 550 | 410 | 45 | 50 | 45 |

## TABLE 2

### FAR ADVANCED WHITE CASES

| Time Under Treatment | No. of Cases | Sputum Positive | Sputum Converted | Cavities Open | Cavities Closed | CLINICAL STATUS | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | Arrested | Apparently Arrested | Quiescent | Improved | Unimproved |
| 6 months | 20 | 20 | 13 | 5 | 9 | .. | .. | 4 | 15 | 1 |
| 6 to 12 months | 54 | 54 | 40 | 16 | 27 | .. | 1 | 13 | 33 | 7 |
| 12 to 18 months | 28 | 28 | 24 | 6 | 20 | 4 | 1 | 7 | 13 | 3 |
| 18 to 24 months | 17 | 17 | 16 | 2 | 11 | 4 | .. | 7 | 5 | 1 |
| 24 to 30 months | 13 | 13 | 11 | 2 | 12 | 4 | .. | 4 | 4 | 1 |
| 30 to 36 months | 2 | 2 | 2 | .. | 1 | 2 | .. | .. | .. | .. |
| TOTAL | 134 | 134 | 106 | 31 | 80 | 14 | 2 | 35 | 70 | 13 |

### MODERATELY ADVANCED WHITE CASES

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| 6 months | 3 | 3 | 3 | .. | 3 | .. | .. | .. | 3 | .. |
| 6 to 12 months | 8 | 8 | 6 | 1 | 3 | 1 | .. | 3 | 2 | 2 |
| 12 to 18 months | 3 | 3 | 3 | .. | 2 | 1 | .. | .. | 2 | .. |
| 18 to 24 months | 2 | 2 | 2 | .. | 1 | 1 | .. | .. | 1 | .. |
| 24 to 30 months | 2 | 2 | 2 | .. | 1 | 1 | .. | 1 | .. | .. |
| 30 to 36 months | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| TOTAL | 18 | 18 | 16 | 1 | 10 | 4 | .. | 4 | 8 | 2 |

## TABLE 3

### FAR ADVANCED NEGRO CASES

| Time Under Treatment | No. of Cases | Sputum Positive | Sputum Converted | Cavities Open | Cavities Closed | CLINICAL STATUS | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | Arrested | Apparently Arrested | Quiescent | Improved | Unimproved |
| 6 months | 11 | 11 | 6 | 2 | 1 | .. | .. | 1 | 9 | 1 |
| 6 to 12 months | 35 | 35 | 23 | 12 | 21 | .. | .. | 14 | 15 | 6 |
| 12 to 18 months | 14 | 14 | 12 | 3 | 4 | .. | .. | 9 | 3 | 2 |
| 18 to 24 months | 10 | 10 | 9 | 1 | 9 | 1 | 1 | 5 | 2 | 1 |
| 24 to 30 months | 3 | 3 | 3 | .. | 1 | 1 | .. | 2 | .. | .. |
| 30 to 36 months | 3 | 3 | 3 | .. | 1 | 1 | .. | 2 | .. | .. |
| TOTAL | 76 | 76 | 56 | 18 | 37 | 3 | 1 | 33 | 29 | 10 |

### MODERATELY ADVANCED NEGRO CASES

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| 6 months | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 6 to 12 months | 2 | 2 | 2 | .. | 1 | .. | .. | 2 | .. | .. |
| 12 to 18 months | 3 | 3 | 3 | .. | 3 | .. | .. | 3 | .. | .. |
| 18 to 24 months | 1 | 1 | 1 | .. | 1 | .. | .. | 1 | .. | .. |
| 24 to 30 months | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 30 to 36 months | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| TOTAL | 6 | 6 | 6 | .. | 5 | .. | .. | 6 | .. | .. |

TABLE 4

FAR ADVANCED WHITE CASES

| Time Under Treatment | No. of Cases | Sputum Positive | Sputum Converted | Cavities Open | Cavities Closed | CLINICAL STATUS | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | Arrested | Apparently Arrested | Quiescent | Improved | Unimproved |
| Less than 6 mos. | 107 | 107 | 45 | 69 | 18 | .. | .. | 1 | 77 | 29 |
| **FAR ADVANCED NEGRO CASES** | | | | | | | | | | |
| Less than 6 mos. | 44 | 44 | 20 | 19 | 9 | .. | .. | .. | 33 | 11 |
| TOTAL | 151 | 151 | 65 | 88 | 27 | .. | .. | 1 | 110 | 40 |
| **MODERATELY ADVANCED WHITE CASES** | | | | | | | | | | |
| Less than 6 mos. | 15 | 15 | 9 | 7 | 4 | .. | .. | .. | 14 | 1 |
| **MODERATELY ADVANCED NEGRO CASES** | | | | | | | | | | |
| Less than 6 mos. | 10 | 10 | 9 | 2 | .. | .. | .. | 2 | 7 | 1 |
| TOTAL | 25 | 25 | 18 | 9 | 4 | .. | .. | 2 | 21 | 2 |

## TABLE 3

### FAR ADVANCED NEGRO CASES

| Time Under Treatment | No. of Cases | Sputum Positive | Sputum Converted | Cavities Open | Cavities Closed | CLINICAL STATUS | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | Arrested | Apparently Arrested | Quiescent | Improved | Unimproved |
| 6 months | 11 | 11 | 6 | 2 | 1 | .. | .. | 1 | 9 | 1 |
| 6 to 12 months | 35 | 35 | 23 | 12 | 21 | .. | .. | 14 | 15 | 6 |
| 12 to 18 months | 14 | 14 | 12 | 3 | 4 | .. | .. | 9 | 3 | 2 |
| 18 to 24 months | 10 | 10 | 9 | 1 | 9 | 1 | 1 | 5 | 2 | 1 |
| 24 to 30 months | 3 | 3 | 3 | .. | 1 | 1 | .. | 2 | .. | .. |
| 30 to 36 months | 3 | 3 | 3 | .. | 1 | 1 | .. | 2 | .. | .. |
| TOTAL | 76 | 76 | 56 | 18 | 37 | 3 | 1 | 33 | 29 | 10 |

### MODERATELY ADVANCED NEGRO CASES

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| 6 months | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 6 to 12 months | 2 | 2 | 2 | .. | 1 | .. | .. | 2 | .. | .. |
| 12 to 18 months | 3 | 3 | 3 | .. | 3 | .. | .. | 3 | .. | .. |
| 18 to 24 months | 1 | 1 | 1 | .. | 1 | .. | .. | 1 | .. | .. |
| 24 to 30 months | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| 30 to 36 months | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| TOTAL | 6 | 6 | 6 | .. | 5 | .. | .. | 6 | .. | .. |

TABLE 4

FAR ADVANCED WHITE CASES

| Time Under Treatment | No. of Cases | Sputum Positive | Sputum Converted | Cavities Open | Cavities Closed | CLINICAL STATUS | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | Arrested | Apparently Arrested | Quiescent | Improved | Unimproved |
| Less than 6 mos. | 107 | 107 | 45 | 69 | 18 | .. | .. | 1 | 77 | 29 |
| **FAR ADVANCED NEGRO CASES** | | | | | | | | | | |
| Less than 6 mos. | 44 | 44 | 20 | 19 | 9 | .. | .. | .. | 33 | 11 |
| TOTAL | 151 | 151 | 65 | 88 | 27 | .. | .. | 1 | 110 | 40 |
| **MODERATELY ADVANCED WHITE CASES** | | | | | | | | | | |
| Less than 6 mos. | 15 | 15 | 9 | 7 | 4 | .. | .. | .. | 14 | 1 |
| **MODERATELY ADVANCED NEGRO CASES** | | | | | | | | | | |
| Less than 6 mos. | 10 | 10 | 9 | 2 | .. | .. | .. | 2 | 7 | 1 |
| TOTAL | 25 | 25 | 18 | 9 | 4 | .. | .. | 2 | 21 | 2 |

cavity closure in 5 cases with cavitation was 100 per cent. All six cases at present are in the quiescent stage.

In this review one may be questioned in presenting the group in Table IV. We realize that the time element is very important in evaluating any form of pulmonary collapse, and many of these cases have had the treatment for only two or three months. In analyzing the table, however, and noting the percentage of sputum conversions and cavity closure, their review seems justified.

There are 151 white and negro cases with far advanced disease. Sputum conversion occurred in 65, or 43 per cent and cavity closure in 27 of 115 cases with cavitation, or 23 per cent. In the moderately advanced group of 25 treated less than 6 months, 18 converted their sputum, or 72 per cent; cavity closure occurred in 4 of 13 cases with cavitation, or 31 per cent.

Sputum studies have been by the concentrate method of 72-hour specimens. If the sputum is found negative, a culture or guinea pig inoculation is done. Examination of gastric washings is done if a satisfactory sputum specimen can not be obtained. Cavity closure is confirmed by planigraphic study if any question arises in the interpretation of the chest x-ray film.

One of the indications for pneumoperitoneum is to prepare the patient for major surgery. Forty-two cases with extensive disease showed such remarkable improvement that thoracoplasty could be done for residual thick-walled cavities at the apex. One lobectomy was done for a mid-zone cavity on the right. Two lucite packs were inserted for residual cavities at the apex, the patients refusing thoracoplasty.

In the group of 50 cases discontinued as unsatisfactory, 31 were white and 19 negro. This seems a rather large number and probably indicates an error in judgment in selection of cases to be treated. All were in the far advanced group, prognosis was poor, and any type of collapse therapy was probably not indicated. Extension of disease under treatment was the cause of discontinuing it in 31 cases. Other conditions and complications developed in 19 cases and influenced the decision to discontinue treatment. Failure of the diaphragm to rise because of adhesions was the cause in 6 cases. Severe intestinal tuberculosis developed in 2 cases. Tuberculosis of the spine, intestinal obstruction and amyloidosis accounted for 3 cases. Two cases had severe endobronchial disease and dyspnea. One of these patients was placed on streptomycin therapy and has improved. Two patients developed a large amount of abdominal fluid, obliterative peritonitis developed, and refills became difficult to give. One of these cases had reached the quiescent stage at the time the fluid appeared. One patient, in whom treatment was discontinued as unsatisfactory, had rectal

and scrotal tuberculous sinuses and progressive pulmonary disease. He was placed on bed rest, streptomycin, and a fistulectomy was done. At the present time all sinuses are closed, his sputum is negative, and he is classified as quiescent.

In the group of 45 fatal cases, 26 were white and 19 negro. In 43, the cause of death was extensive pulmonary tuberculosis. One patient died of coronary occlusion, phlebothrombosis of the left leg and pulmonary embolism. A pericardial effusion developed in one case. A pericardial tap was done and the fluid on examination revealed tubercle bacilli.

The 410 cases still under treatment have been classified as to age. In the age group of 20 to 30 years we find 235 cases, 89 in the group 30 to 40 years, 40 in the group 40 to 50 years, and 46 in the oldest age group of 50 to 60 years.

Phreniclasia was successfully done on the right in 194 cases and on the left in 166. There were 116 unsatisfactory pneumothoraces discontinued and the lungs reexpanded. Pneumoperitoneum was then instituted with satisfactory results. Pneumoperitoneum was given in combination with pneumothorax in 7 cases, and in 10 cases with thoracoplasty completed pneumoperitoneum was added to control a spread to the contralateral side. There were 76 cases found with endobronchial tuberculosis. This condition was treated with the application of 30 per cent silver nitrate and the pneumoperitoneum continued.

### *Discussion*

The ultimate aim of any form of treatment is to aid in the recovery of the patient, and after reviewing the results obtained in this series, we are convinced that pneumoperitoneum is of great value in the treatment of pulmonary tuberculosis. It should not be considered a method of treatment to replace all other types of collapse therapy, but another procedure to be used by the physician in selected cases. Ill-chosen or indiscriminate administration of the procedure is bound to discredit the treatment.

In properly selected cases, pneumoperitoneum, when given at regular intervals, is capable of inducing good drainage and relaxation of the lung. These two outstanding factors are the reasons for the excellent results seen in this series. This therapy appears most logical in the treatment of bilateral disease, although the selection of more favorable cases found the results more impressive. We have found this method of treatment capable of effecting a cure in many patients who unquestionably were not suitable for either pneumothorax or thoracoplasty. The procedure has also made suitable for thoracoplasty a considerable number of cases not likely to become so without some form of preliminary collapse.

If indications for pneumothorax are present, we feel that this type of collapse should be instituted. The use of pneumoperitoneum, however, has the following advantages over the use of artificial pneumothorax:

1) The disease in the lungs can be observed at all times by fluoroscopy and roentgenography and its response to therapy. This cannot be done in a large percentage of pneumothorax cases, since the diseased portion is so collapsed that it cannot be followed without reexpansion.

2) The complications are extremely few. Thickened pleura, spontaneous collapse, empyema, and non-expandable lungs are seldom seen.

3) Pneumoperitoneum may be discontinued or continued again far more readily than artificial pneumothorax.

4) Pneumoperitoneum treats bilateral disease at the same time, providing a great saving of time to both patient and physician.

The treatment of pulmonary tuberculosis with pneumoperitoneum is going through the same stages as experienced with pneumothorax. There is a tendency to give too large refills as was done in the early days of pneumothorax therapy. We are endeavoring to secure the maximum lift of the diaphragm, and excessive refills merely depress the abdominal contents downward and toward the spine, and the patient is made uncomfortable. In our experience, the average refill after the pneumoperitoneum is well established should be 900 cc. of air weekly, and the intra-abdominal pressure built up to a positive pressure of 10 cm. of water. Cases with a lift of the diaphragm to the anterior third rib secure the best result.

Pneumothorax therapy in the treatment of pulmonary tuberculosis in the negro has been unsatisfactory in this hospital. Complications have resulted, though the cases have been handled carefully with frequent small refills, and the patient given the benefit of early pneumonolysis if adhesions were present. In acute bilateral disease the introduction of pneumoperitoneum has produced excellent results. In the past, disease of this type in the negro has been looked upon as fatal, and such a result could not have been obtained by any other method of collapse therapy at our disposal. We feel that pneumoperitoneum is the treatment of choice in the negro.

Following the termination of collapse there is a maximum and satisfactory restoration of the normal lung volume and function provided a permanent paralysis of the hemidiaphragm has not occurred. When treatment is terminated, refills are simply discontinued, and no undesirable effects have been observed.

The criteria upon which we must base our appraisal of the value

of pneumoperitoneum is its ability to close cavities, convert the sputum and heal the lung with a minimum of serious complications. We have tried it in a large series of white and negro cases and find that it meets this criterion.

## SUMMARY

1) A therapeutic review of 550 patients with pulmonary tuberculosis, treated by a combination of temporary phrenic nerve interruption and pneumoperitoneum, has been presented.

2) The physiology, indications, and complications have been stated.

3) The technique of the procedure of pneumoperitoneum has been outlined.

4) It is of value in preparing the patient for thoracoplasty.

5) This method of treatment is capable of effecting a cure in many cases which unquestionably are not suitable either for pneumothorax or thoracoplasty.

6) Cavity closure occurs equally as well at the apex as in the basal portion of the lung.

7) In 134 far advanced white patients treated for six months or more, 106, or 79 per cent converted their sputum. Cavity closure occurred in 80 cases, or 72 per cent. In the far advanced 76 negro patients, 56 or 73 per cent converted their sputum; cavity closure occurred in 37, or 67 per cent. In the more favorably selected cases, the results were more impressive.

8) The duration of treatment parallels that required by other types of collapse therapy.

9) The importance of maximum elevation of the diaphragm is emphasized.

10) Artificial pneumoperitoneum combined with phrenic crush has been found a valuable treatment; it avoids the serious complications of artificial pneumothorax and gives to many patients, hopelessly ill, their chance to recover.

## RESUMEN

1) Se ha presentado un repaso terapéutico de 550 tuberculosos pulmonares tratados mediante la combinación de la interrupción temporal del nervio frénico y neumoperitoneo.

2) Se han expuesto la fisiología, las indicaciones y las complicaciones.

3) Se ha bosquejado la técnica del neumoperitoneo.

4) Es valioso para preparar al paciente para la toracoplastia.

5) Este método de tratamiento es capaz de efectuar curaciones en muchos casos que indudablemente no son apropiados para el neumotórax o la toracoplastia.

6) Cierres de cavernas tienen lugar igualmente bien en el vértice o en la zona basal del pulmón.

7) En 134 pacientes blancos muy avanzados, tratados por seis meses o más, en 106, el 79 por ciento, se convirtió el esputo. Cierre de cavernas ocurrió en 80 casos, o sea en el 72 por ciento. En los 76 pacientes negros muy avanzados, en 56, el 73 por ciento, se convirtió el esputo; cierre de cavernas ocurrió en 37, o en el 67 por ciento. En los casos seleccionados más favorablemente los resultados fueron aún más imponentes.

8) La duración del tratamiento paralela el requerido en otros tipos de colapsoterapia.

9) Se hace hincapié sobre la importancia de obtener la elevación máxima del diafragma.

10) Se ha encontrado que el neumoperitoneo artificial combinado con la trituración del frénico es un tratamiento valioso que evita las graves complicaciones del neumotórax artificial y que les ofrece a muchos pacientes desesperanzados la oportunidad de recobrar la salud.

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## Discussion

LEON H. HIRSH, M.D., F.C.C.P.
Milwaukee, Wisconsin

I should like to compliment Dr. Moyer on the excellence and the completeness of his study.

Pneumoperitoneum has justly assumed its place in our armamentarium against tuberculosis. I have been using therapeutic pneumoperitoneum for the past ten years. With reference to the safety and simplicity of the procedure, I wish to mention that I have established pneumoperitoneum in my office. Also, patients who have been discharged from the sanatorium are maintained as ambulatory pneumoperitoneum subjects in the office, and present no greater problem than those receiving ambulatory pneumothorax treatment.

Concerning the site of injection of the air, I prefer to introduce the initial and a few subsequent inflations in the left lower quadrant of the abdomen. However, when there is an adequate rise of the diaphragm, I usually choose a site through the lower thorax. I do not insist on the attainment of a set positive pressure. I prefer to gauge the amount of air according to the position of the diaphragm and the reaction of the patient. For subjective reasons on the part of the patient, it is expedient to use local anaesthesia for each refill.

I have found it advantageous and entirely safe to have an

assistant attach a rubber bulb to the distal bottle of a Robinson pneumothorax apparatus and pump the air into the peritoneal cavity rather rapidly.

I should like to add the following to Dr. Moyer's list of indications for pneumoperitoneum in pulmonary tuberculosis: (1) Patients following childbirth are likely to suffer with exacerbation of their pulmonary lesions. Here, pneumoperitoneum, when induced after delivery, maintains the diaphragm at the high level of the last trimester of pregnancy, and continues the pulmonary relaxation which would otherwise be lost. (2) Elderly individuals who are not good bilateral pneumothorax prospects will often do well with pneumoperitoneum provided there is no severe cardiovascular impairment. (3) Patients who have tuberculous enterocolitis in addition to pulmonary lesions will obtain multiple benefits from this procedure. (4) Individuals who suffer with thoracic pain due to basal diaphragmatic adhesions will enjoy some relief with pneumoperitoneum.

As Dr. Moyer has said, one may anticipate good results from therapeutic pneumoperitoneum only if it is employed in critically selected instances. Then one should allow a sufficient length of time to achieve healing and recovery with the use of this measure. Consider the time of healing in the natural reparative processes of tuberculosis—particularly that type which is selective for pneumoperitoneum. Patients who have had thoracoplasty may not be expected to convert their sputum in possibly one to two years, and even many continue under the classification of "maximum benefit." One is not justified, therefore, in discontinuing artificial pneumoperitoneum if conclusive therapeutic results, roentgenologic or bacteriologic, are not accomplished in two or three months. This commentary does not apply to the study just presented. However, with the prevailing views toward short term pneumothorax there is a popular tendency to evaluate pneumoperitoneum in the same light. While the fundamental biologic processes are similar, I feel that by the very nature of the type of disease selected for artificial pneumoperitoneum one should not expect dramatic therapeutic response in too short a time.

There is another point of importance which I wish to mention. It is well known that following reexpansion of the lung after artificial pneumothorax there remains in most instances some degree of functional impairment of the previously relaxed lung due to plural changes. Since pneumoperitoneum does not influence the pleurae, this treatment is free of the liability of producing a post-therapeutic pulmonary insufficiency.

I wish to congratulate Dr. Moyer again on his splendid presentation of this very important subject.

# Discussion

JACOB J. MENDELSOHN, M.D., F.C.C.P.
Chicago, Illinois

The controversial aspects of pneumoperitoneum with phreniclasia in the treatment of chronic pulmonary tuberculosis are in a great measure clarified by Dr. Moyer's interesting and instructive presentation. His wealth of clinical material and his striking portrayal of the subject offers convincing testimony in support of his conclusion that there are definite indications for this type of collapse therapy.

Based upon Dr. Banyai's studies on the subject we have employed pneumoperitoneum with phreniclasia in selected cases over a period of several years with clinical observations similar to those presented by Dr. Moyer. Pneumoperitoneum often aids the action of phrenic nerve paralysis and favorable results have been noted in apical cavities as well as in lesions of the lower lobes. In the treatment of tension cavities where artificial pneumothorax is inapplicable or unsatisfactory the percentage of closure following pneumoperitoneum with phrenic paralysis compares favorably with those obtained with the more radical methods of collapse therapy.

The following case demonstrates the possibilities of this type of treatment: This patient, age 47 was admitted to Fox River Sanitarium in 1942 with a diagnosis of far advanced pulmonary tuberculosis complicated by tuberculous enteritis. His sputum was positive for tubercle bacilli. Figure 1 shows a tension cavity de-

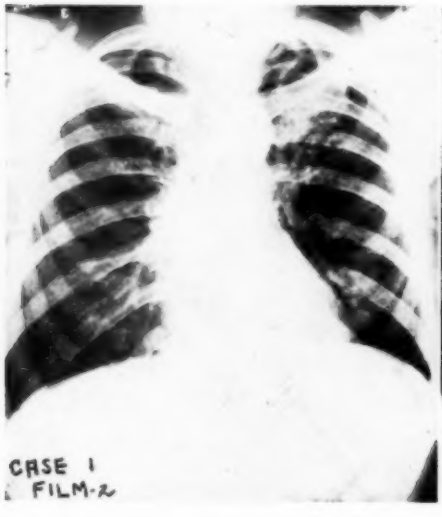


FIGURE 1

FIGURE 2

veloping during the course of pneumothorax treatment. Pneumothorax was discontinued and Figure 2 reveals closure of the cavity following phreniclasia with pneumoperitoneum. He is now an arrested case and is employed eight hours a day as a salesman.

Dr. Moyer should be complimented for his valuable contribution to the progress of tuberculosis therapy. It has been a privilege to discuss his excellent paper.

# Differential Diagnosis and Treatment of Congenital Cystic Malformation of the Lung*

W. E. ADAMS, M.D.
Chicago, Illinois

Congenital cystic malformation of the lung presents an interesting and difficult problem in differential diagnosis since the clinical picture frequently is very similar to that produced by a number of other conditions within the thorax. Pulmonary cystic lesions may be of two types: congenital and acquired. This paper has to deal only with the former type.

The symptomotology produced by congenital malformation of the lung is brought about by one of two mechanisms; i.e. (1) disturbance of intrathoracic pressure relations due to overdistension of the pulmonary cyst, and (2) symptoms of pulmonary suppurative disease following the development of infection of the cyst.

In spite of the recognition of the pitfalls in diagnosis as reported by various authors[1-4] the differentiation from a number of intrathoracic lesions frequently presents a difficult problem. The importance of establishing a correct diagnosis before operation will be brought out strikingly in the cases to be presented. Many of these demonstrate long morbidity and incapacitation or hospitalization for treatment of a condition other than the one to be discussed.

The present study is based on experience with thirty patients, twenty-seven of which had symptoms following the development of infection of the malformed area. In the remaining three, symptoms were produced when intrathoracic pressures became markedly disturbed through overdistension of the cyst. The age of the patients range from a few days to twenty-five years with the exception of one patient who was thirty-eight and another who was fifty-nine years. The lesions were fairly evenly distributed among the various lung lobes. In no case was there bilateral involvement. In a review of twenty-six reported cases by nine authors, the age distribution, duration of symptoms and location of lesions were approximately the same as in the present series.[5-13]

*Pathology:* The gross pathologic picture in the thirty cases studied was classified as follows: (1) those which presented multiple cysts and considerable functioning lung; (2) those with

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multiple cysts but with little or no normally developed lungs; (3) those which presented a large amount of abnormally developed bronchial tree elements, a number of smaller cysts and little or no normally developed terminal respiratory units. This is an arbitrary classification based on the degree and character of involvement. The records of patients presented illustrate that at times this factor had considerable influence on the clinical course as well as the overall pathologic picture.

Microscopically viewed, the cysts were lined by columnar ciliated epithelium except when as a result of metaplasia it had been replaced by squamous epithelium or had been entirely destroyed by infection. The cysts were surrounded by a variable amount of fibrous connective tissue with bronchial wall elements such as cartilage, glands, and smooth muscle being present in some regions. Outside of the cystic areas many small air passages lined by cuboidal or low columnar epithelium were present. The amount of this type of abnormal development varied considerably in the sections of different patients. When infection had been present throughout the lung, the usual changes characteristic of pneumonitis, such as round cell infiltration, macrophages, and plasma cells, were present. The influence of diffuse chronic pneumonitis on the clinical picture as well as the diagnostic problem will be discussed in more detail under that heading.

The diagnosis of congenital cystic disease of the lung is usually not difficult when its possibility is kept in mind. In those cases where symptoms are produced by overdistension of the cyst with air, the conditions most frequently confused are spontaneous pneumothorax and diaphragmatic hernia. When fluid is present, empyema is suspected. When the patient is seen following the development of infection, lung abscess, bronchiectasis, tuberculosis, pneumonitis and chronic bronchitis are frequently diagnosed before the true nature of the lesion is discovered. In the present study the following conditions were diagnosed previous to recognition of the lesions: (a) bronchiectasis, 17; (b) lung abscess, 10; (c) empyema, 5; (d) tuberculosis, 5; (e) pneumothorax, 3; (f) pneumonitis, 3; (g) diaphragmatic hernia, 3; (h) chronic bronchitis, 1. In the 26 reported cases, a similar group of conditions was considered in differential diagnosis. In spite of the various diagnostic methods for studying these lesions, it is frequently extremely difficult to differentiate between one of the above lesions and that of cystic malformation of the lung. A careful history of the onset of the clinical course together with x-ray films of the chest and information from bronchoscopic examination will usually lead to a correct diagnosis in a high percentage of cases when the possibility of the lesion is kept in mind. The

following case reports are representative of difficulties encountered in making a diagnosis.

*Lung Abscess:* 1) An 18-year-old white male complained of having had pneumonia at the age of five years. Following this, a lung abscess developed and was drained through the chest wall. After a period of time the drain was removed. The symptoms of productive cough and sepsis returned, however. The abscess cavity was again drained and allowed to close on three successive occasions. The last drainage tube had been in place for approximately twelve years when the patient was first seen in this clinic. During this period he had developed normally and had no complaints other than the presence of the draining sinus.

Physical examination revealed a healthy appearing obese boy with a large tube draining a sinus at the base of the right lung posteriorly. On removing the tube, a smooth lined cavity approximately two to two and one-half inches in diameter was visible. Several bronchial fistulae were demonstrable. X-ray films following injection of the sinus with iodized oil showed a fluid level just below the dome of the diaphragm. The diagnosis of congenital cystic malformation of the lung was made and the right lower lobe was resected. The postoperative course was uneventful.

The surgical specimen showed a large cyst involving approximately one-third of the lower lobe, the remainder of the lobe being largely airless. A microscopic section through the larger portion of the lobe showed much of the lung abnormally developed. The cyst was lined with columnar ciliated or stratified squamous epithelium.

2) A 38-year-old white female was admitted to the clinic complaining of a productive cough with foul sputum of approximately one year's duration. Four weeks prior to the onset of her symptoms a tonsillectomy had been made. She had been treated with penicillin and streptomycin which gave temporary relief and at one time a thoracentesis was said to yield frank pus. She had lost thirty pounds in weight.

TABLE 1: CONGENITAL CYSTIC MALFORMATION OF THE LUNG DIFFERENTIAL DIAGNOSIS

| | Authors | Reported |
|---|---|---|
| 1. Bronchiectasis | 17 | 4 |
| 2. Lung Abscess | 10 | 2 |
| 3. Empyema | 5 | 4 |
| 4. Tuberculosis | 5 | 2 |
| 5. Pneumothorax | 3 | 1 |
| 6. Pneumonitis | 3 | 0 |
| 7. Diaphragmatic Hernia | 3 | 0 |
| 8. Chronic Bronchitis | 1 | 0 |
| 9. Pleuropericardial cyst | 0 | 1 |
| 10. No diagnosis | 0 | 11 |
| No. Cases in Each Series | 30 | 26 |

On admission she appeared chronically ill, had a fever of 38° C. and a leukocytosis of 16,700. The hemoglobin was 11.5 grams and the red count 3.7 million. The positive physical findings were limited to the right chest which showed some decrease in tactile fremitus and a few moist rales in the mid-lung field posteriorly. The sputum raised by the patient was extremely foul, quite thick and greenish-yellow in color. It contained a variety of organisms commonly found in pulmonary suppurative lesions. There were no acid-fast organisms although the patient had been suspected of having had tuberculosis and was kept in a sanatorium for approximately two weeks. An x-ray examination revealed a fluid level containing cavity with a relatively small amount of infiltration surrounding, located in the right mid-lung field posteriorly. The diagnosis was nontuberculous lung abscess. Bronchoscopic examination showed much foul thick pus coming from the right lower lobe.

The patient was prepared for operation by chemotherapy and blood transfusions and the right lower lobe was removed. The upper lobe appeared normal. Her convalescence was entirely uneventful. The surgical specimen when opened showed a large cavity in the upper part of the right lung posteriorly. A moderate amount of fibrosis was present about the cavity. The lower portion of the lung appeared normal. A microscopic section made through the entire wall of the cavity revealed an abnormally developed lung with the cavity lined by columnar ciliated epithelium except in some places where it had been replaced by squamous epithelium.

The diagnosis of lung abscess in this patient was a very natural one in view of the onset of a productive cough a short time following tonsillectomy, since that operation under anesthesia is one of the more frequent causes of lung abscess. When the lesion did not decrease in size under medical management, in spite of the clinical picture being markedly improved, a suspicion of malformation of the lung should have been entertained. This was especially true when the patient was first seen in this clinic a year following the onset of her illness. A large pulmonary cavity presenting a fluid level, unaccompanied by sepsis or a positive sputum for tubercle bacilli, is most likely to be a congenital lung cyst.

In patients having chronic pneumonitis surrounding cystic areas, the diagnosis of lung abscess is a common error. In such cases after drainage of the cavity the space does not become obliterated. If a biopsy is not taken, the true nature of the disease may remain unrecognized and a number of operations may be made in trying to heal the lesion. In such cases the history should arouse one's suspicion of the possibility of the condition. When the correct diagnosis is made, operative treatment should consist of excision rather than drainage unless contraindicated by the patient's general condition. Next to bronchiectasis, lung abscess has been diagnosed most frequently in the present series of cases prior to recognition of the true nature of the lesion.

*Bronchiectasis:* P.K., a white female 25 years of age, complained of a productive cough with intermittent attacks of fever over a period of twenty-four years. She had had whooping cough at the age of one year and had developed pneumonia during her second pregnancy. Examination revealed a poorly nourished, chronically ill individual who coughed considerably during the procedure. Many rales were heard in both bases. X-rays following iodized oil injection showed the characteristic picture of bilateral bronchiectasis. On bronchoscopy a purulent exudate was seen coming from the bronchus of all lobes. A diagnosis of bilateral bronchiectasis was made and the patient was prepared for operation. A resection of the right lower and middle lobes was carried out by the dissection technic. Her postoperative course was uneventful. Her symptoms were decreased to approximately one-third of the original severity and her general condition was good. Examination of the surgical specimen showed the lesion in the lower lobe to be saccular bronchiectasis. However, the right middle lobe was found to be made up entirely of cystic areas. Microscopic sections of this lobe showed the characteristic picture of cystic malformation of the lung. Approximately seven months following the above operation the left lower lobe and lingula were resected. The patient made a good recovery and has been largely relieved of her symptoms.

This patient presented the usual history and findings of bilateral bronchiectasis, the true nature of the lesion in the middle lobe being found only after the surgical specimen was examined. One might speculate as to whether infection of the cyst in the middle lobe had led to the development of bronchiectasis in the lower lobes due to repeated contamination by internal drainage.

In three other patients of the group under study, the middle lobe was involved by the malformation. In these cases there was no infection in the lower lobes and on iodized oil injection pathological changes were seen only in the middle lobe. From our experience with the present group of cases it would seem that when only the middle lobe is involved by a suppurative process, cystic malformation should be suspected rather than primary bronchiectasis. In patients with middle lobe involvement, internal drainage of secretions into the lower lobes may produce a picture on bronchography suggestive of tubular bronchiectasis and thus lead to the resection of normal lung tissue. At times it may also suggest that the bilateral involvement is too severe for operation to be advisable. In the three above mentioned patients, the middle lobe was resected followed by complete relief of symptoms.

*Chronic (Diffuse) Pneumonitis:* 1) An eight-year-old white male was first seen complaining of a productive cough with intermittent hemoptysis and fever of approximately one year's duration. The patient gave a history of bilateral pneumonia with surgical drainage of bilateral empyema during the earlier part of this period. On examination he appeared chronically ill and was somewhat dyspneic. The entire left lung was dull to flat, and breath sounds were either absent or bronchial

in character. The right lung was normal. Laboratory findings included hemoglobin, 10 gms.; red blood count, 3.9 million; and white blood count, 14,000. X-ray films showed the left lung to be entirely airless with a slight deviation of the heart shadow toward the right side. Following the injection of iodized oil, only the primary bronchi of the upper and lower lobes were outlined. On bronchoscopic examination a moderate amount of foul pus exuded from the left main stem bronchus and an especially large amount from the left lobe. The right side was clear. The differential diagnosis included massive empyema, chronic suppurative pneumonitis with abscess formation, and possibly bronchiectasis. Pneumonitis, however, was thought to be the most likely cause of his trouble. A thoracentesis revealed no evidence of pus in the pleural cavity. Therefore, after preparation of the patient for operation by several transfusions agregating a total of 900 cc. of whole blood and postural and bronchoscopic drainage as well as chemotherapy, an exploration was made. The left upper lobe was found to be completely atelectatic but not infiltrated or otherwise abnormal. The left lower lobe was diffusely infiltrated and airless, but no large cystic spaces could be identified. The lower lobe was removed by the dissection technic, and although the upper lobe could not be re-expanded, it was not resected.

Following operation the patient made an uneventful recovery. The upper lobe remained completely atelectatic for a period of approximately four to six weeks then became inflated spontaneously.

On cut section the surgical specimen revealed several cavities filled with frank pus. The remainder of the tissue consisted of markedly fibrosed and consolidated lung. On microscopic examination the walls of the spaces were lined by columnar ciliated or stratified squamous epithelium, and various elements of the bronchial wall were present. The remainder of the lung showed abnormal development of the various lung elements, numerous small spaces lined by cuboidal epithelium and much fibrosis. There was a complete lack of normally developed terminal respiratory units. The degree of infection was not as great as suspected from gross examination of the tissue.

2) A 10½-year-old female entered the hospital with a diagnosis of chronic lung abscess of two years duration. Two weeks prior to her pulmonary symptoms, her tonsils had been removed under general anesthesia. Following this she began to have a productive cough of green purulent sputum, associated with fever and other symptoms of sepsis. After treatment by conservative methods for one year, a cavity in the lower part of the right lung was drained with a tube. The patient has continued to drain from this area since and one several occasions there have been mild hemorrhages. She has lost eight pounds in weight and tires easily. There has been no exposure to tuberculosis.

Examination revealed a poorly nourished, chronically ill girl who was running a low grade fever. The leukocyte count was 8,900; hemoglobin, 11.3 grams and RBC, 3.7 million. Except for clubbing of the fingers, the positive physical findings were limited to the thorax where a rubber tube drain was seen in the 7th right interspace. Expansion of the right chest was markedly limited and tactile fremitus diminished. Breath sounds over the right chest was entirely absent and the percussion note was flat. X-ray films of the chest showed a diffuse opacity involving the entire right side with what appeared to be a cavity formation in the lower portion. The left lung was clear. The diagnosis was lung abscess

with questionable empyema. Bronchoscopic examination revealed some pus coming from the right main bronchus.

The patient was prepared for operation by chemotherapy, supportive measures including blood transfusions. On exploration the entire right lung was found to be infiltrated and airless. Because the lesion involved the entire lung it was thought best to do a pneumonectomy. For the first two weeks after operation the patient had an uneventful convalescence, but evidence of empyema developed at that time. This was drained and subsequently a thoracoplasty was performed to obliterate the space. The patient has remained asymptomatic since.

On opening the surgical specimen one large, multi-loculated cavity was found involving a large portion of the lung. The remainder of the lung was airless, infiltrated and fibrotic. A microscopic section made through the entire lesion revealed the lining of the cavity to be columnar ciliated epithelium except where it had been replaced by squamous epithelium. Outside of the main cavity other evidence of malformation of the lung was present.

In patients with a pathological process such as the above described, it is frequently impossible to differentiate between a chronic pneumonitis and other pathologic lesions. A history of insidious onset of a pulmonary suppurative process in a child without the usual etiologic factor for the production of bronchiectasis or lung abscess should strongly suggest the possibility of congenital cystic disease. Bronchoscopic examination and x-rays following iodized oil injection are usually not diagnostic. In such cases when the patient's condition warrants it and a thorough preparation for operation has been made, exploration and resection of the involved portion is indicated.

*Empyema:* A white female, 2½ years of age, was admitted complaining of some difficulty in breathing and a productive cough of two months' duration. She had also had some elevation of temperature during this period. Examination revealed an underdeveloped and undernourished sickly child whose right chest was dull to flat over the lower three-fourths of the lung. A few rales were heard at the apex. Breath sounds were diminished or absent over the greater portion of the right side. The white count was nineteen thousand. X-ray films of the chest revealed complete opacity in the lower two-thirds of the right lung. An empyema was then diagnosed and drainage was recommended. At operation after resecting a piece of rib the cavity was entered. However, the wall did not appear to be that of an inflamed pleura; thus, a biopsy was taken. This showed the condition to be a large cyst of the right lower lobe lined by columnar ciliated epithelium. After the cyst had drained for a period of approximately two years, resection of the right lower lobe was carried out without event.

As pointed out by Maier and Haight[1] large infected cysts of the lung are commonly erroneously diagnosed as empyema. In some cases x-rays following diagnostic pneumothorax may be helpful in differential diagnosis in that the limits of the cavity may appear to be more rounded than is usually the case in empyema. Also at

operation the tissue outside of the cavity does not appear to be inflammed in that it is less rigid than is the case in the wall of an empyema. A routine biopsy of the wall is usually diagnostic. Since this diagnosis is usually made in infants or small children, drainage of the cyst prior to resection may frequently be the treatment of choice. However, a correct diagnosis is necessary for determining prognosis and the planning of further care. In most reported cases the diagnosis has been made following drainage of the lesion, and resection of the lung has been made at a later date.

*Pulmonary Tuberculosis:* A 50-year-old colored male complained of a productive cough and fever of several years' duration. There was no history of night sweats or hemoptysis. On some occasions he had become dyspneic and had experienced pain in the left chest. Examination revealed a chronically ill-appearing individual who coughed considerably during the procedure. Many coarse rales were heard over the left chest, and bronchial breathing was marked in the lower half posteriorly. X-ray films showed marked opacity with cavity formation in the lower three-fourths of the left lung. Following iodized oil injection a large cavity was demonstrable in the lower left lung and a number of smaller loculations in other parts of the same side. Examination of the sputum showed several organisms which appeared like tubercle bacilli, and for a time pulmonary tuberculosis was diagnosed. Subsequent examinations of the sputum were negative for tubercle bacilli, however, and drainage of the lower cavity was planned. The patient's condition improved, and the operation was postponed. Because of the patient's age and general disability, resection of the involved lung was thought inadvisable. He has been seen at infrequent intervals during the past nine years and has continued to have a negative sputum for acid fast organisms and has a moderately productive cough but without causing undue handicap. The final diagnosis was congenital cystic malformation of the left lung.

Five of the present series of cases and two of the reported cases had been treated for pulmonary tuberculosis before the true nature of the lesion was recognized. When the possibility of cystic malformation is kept in mind, however, this error in diagnosis is much less likely. Three of the patients had been seen a number of years ago before congenital cystic malformation of the lung was well known. In patients having a productive cough with x-ray evidence of a cavity presenting a fluid level, a repeatedly negative sputum for tubercle bacilli should rule out the diagnosis of pulmonary tuberculosis.

*Spontaneous Pneumothorax:* A 3-year-old colored female was admitted complaining of a mild productive cough, fever and dyspnea of one month's duration. Shortness of breath had not been extreme at any time, and the amount of sputum raised was rather small. Examination revealed a normally developed child of three who did not appear ill. Positive findings were limited to the chest. Rales were heard in both lungs and there was dullness in the lower part of the right side. Breath

sounds were diminished or absent over the upper two-thirds of the right lung. On x-ray examination the right lung appeared to be almost completely collapsed by a spherical shaped space filled with air. The mediastinum was not shifted. The diagnosis was spontaneous pneumothorax. Further examination revealed no evidence of other lesions, and the tuberculin test was normal. Manometric readings showed approximately atmospheric pressures within the air space, the pressure decreasing on inspiration and increasing on expiration. After removing several hundred cubic centimeters of air from the space, the pressures were unchanged. Fluoroscopic examination showed the space to be the same size as before the aspiration.

On closer inspection of the x-ray films it was seen that the outline of the air-filled space was oval in shape. Both at the apex and at the base there was a continuous border encircling this ovoid space. This suggested that the space was within the lung with a very thin shell of lung surrounding it. Therefore, a diagnosis of congenital cystic malformation of the right lung was made.

The patient has been seen at infrequent intervals during the subsequent ten years, and her condition has remained essentially the same. Operative removal of the cyst has been advised and rejected.

Pressure symptoms produced by large cysts of the lung usually occur in young children or infants. In former years a number of these cases have probably been diagnosed as spontaneous pneumothorax, and when complete examinations were not made the true nature of the condition went unrecognized. Four of the present series studied, whose symptoms were produced by intrathoracic pressure changes, were three years of age or less, one being only twelve days old. The x-ray appearance of a spherical outline is highly suggestive of the presence of a large cyst, but is not always conclusive. If the condition persists unchanged for a long period of time, cystic malformation of the lung is the most probable diagnosis. This can usually be determined by establishing a pneumothorax, thus demonstrating the cyst wall, or by the injection of an opaque media such as iodized oil into the cystic space and visualizing the outline of the cyst wall and trabeculae on x-ray examination.

*Chronic Bronchitis:* A 16-year-old white female was first seen complaining of repeated attacks of an upper respiratory infection which produced a chronic cough. This had not kept her from attending school and had not impaired her general health to any considerable degree. Physical examination revealed a well-developed and nourished individual who did not appear to be very ill. The general physical examination as well as that of the chest was entirely normal. Fluoroscopy showed no abnormal findings. A diagnosis of chronic bronchitis was made, and the patient was treated with sulfa drugs and cough medicine. She was seen at infrequent intervals and continued about the same with intermittent exacerbations of her upper respiratory infection. Approximately one year later during a recurrence of her symptoms, an x-ray picture was made which showed two or three spherical cavities demonstrating

a fluid level and located in the lower right lung field. On physical examination a few rales were heard in the right base. The diagnosis was congenital cystic malformation of the right lung. At operation the condition was found to be confined to the right lower lobe which was removed by the dissection technic. The patient made an uneventful recovery and has been asymptomatic during the two year interval since operation.

The surgical specimen contained two large and several small multiloculated cystic spaces which were almost completely surrounded by normally developed and inflated pulmonary tissue. Microscopic examination revealed columnar ciliated and stratified squamous epithelium lining the cystic spaces. A considerable amount of fibrous tissue and some abnormally developed respiratory passages were present in the walls of the cyst. However, approximately one-half to two-thirds of the lung parenchyma appeared to be normally developed.

The above case illustrates that cystic areas may be easily overlooked both on physical and fluoroscopic examination when little or no secretion is present and where normally functioning lung tissue surrounds the cyst. Furthermore, when the cysts are located below the level of the dome of the diaphragm posteriorly, they are not demonstrable on antero-posterior x-ray views of the chest. A mild cough with little sputum may be the only symptoms present. The condition may go unrecognized until infection increases the fluid contents of the cysts. A history of continued cough in children or young adults without obvious cause should be looked upon with suspicion and repeated fluoroscopic and x-ray examination made for evidence of the lesion.

Treatment consists of resection of the involved part. The risk of operation is small when the patient is properly prepared. In the present group of thirty patients there was one pneumonectomy, one bilateral resection, two bilobectomies. The remainder had one lobe removed. Complete relief of symptoms followed removal of the diseased part. There were no deaths.

It has thus been shown that many conditions of the chest may be clinically simulated by cystic malformation of the lung. In addition to those illustrated by case presentation, bronchogenic carcinoma located in the periphery of the lung may sometimes be confused with this condition. A fluid containing cyst may present a circumscribed opacity on x-ray examination not dissimilar to that produced by carcinoma. Both the cyst and the carcinoma may for several months remain asymptomatic and be discovered only on routine fluoroscopic or radiographic examination. The differential diagnosis is usually made on the basis of the age of the patient and the x-ray appearance of the lesion. If the patient is under 30 years of age, the lesion is most likely to be a cyst of the lung. On x-ray examination a bronchogenic carcinoma is more apt to have less sharply defined borders and be less spherical

shaped than a cyst. In some cases a definite diagnosis cannot be established without exploration. This is indicated in either case because resection of the lesion is indicated in both.

Three patients in this study were seen in consultation with Dr. M. M. Shaw and Dr. Ed Bryant and were treated at the Provident Hospital.

## SUMMARY

A review of thirty cases of cystic malformation of the lung revealed that a number of pathologic conditions had been diagnosed before the true nature of the lesion was recognized. In several instances treatment had been instituted for one or more of these lesions for periods of over a year. In twenty-six collected cases reported by nine authors, a similar experience was noted. The difficulties in differential diagnosis were based chiefly on the similarity of the clinical course with that of other pathologic conditions of the chest. Although radiographic and fluoroscopic examination remain the best diagnostic methods for differentiation, they are not infallible and must be correlated with the clinical findings. The pathologic diagnosis is important both for an accurate evaluation of the disease and for proper surgical treatment. Since the risk of operation is small, resection of the involved lung is the treatment of choice.

## RESUMEN

Un repaso de treinta casos de anomalías quísticas del pulmón reveló que se habían diagnosticado varios otros estados patológicos antes de que se reconociera la vardadera naturaleza de la lesión. En algunos casos se habían instituído tratamientos para una u otra de estas lesiones por períodos de más de un año. En veinte y seis casos compilados, sobre los cuales informaron nueve autores, se notaron experiencias semejantes. Las dificultades encontradas en el diagnóstico diferencial se debieron principalmente a la semejanza del curso clínico al de otros estados patológicos del tórax. Aunque el examen radiográfico y radioscópico todavía es el mejor método para el diagnóstico diferencial, no es infalible y debe ser correlacionado con los hallazgos clínicos. El diagnóstico patológico es importante tanto para apreciar la enfermedad correctamente como para escoger el tratamiento quirúrgico apropiado. Ya que la operación acarrea poco riesgo, la resección del pulmón afectado es el tratamiento de elección.

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# Pulmonary Embolism: A Precipitating Factor of Acute Coronary Insufficiency*

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Cardiac involvement following pulmonary embolism has been so commonly attributed to acute cor pulmonale that the fact that the left ventricle may be affected as often as the right ventricle has been overlooked. It should be recognized that not only does coronary insufficiency frequently develop following acute massive pulmonary embolism but that it may be the predominant cause of the cardiac sequelae, morbidity and mortality.

Acute coronary insufficiency results from disproportion between the nutritional requirements of the myocardium and the coronary blood flow. In previous publications[1,2] we have presented the concept of acute coronary insufficiency as a complete clinical entity with distinct etiology, pathological physiology, characteristic anatomic, electrocardiographic and clinical features, and treatment. Acute coronary insufficiency may be the result of relative insufficiency of coronary flow due to increased cardiac work, or of absolute diminution of coronary flow, or of anoxemia occurring with a normal coronary flow. In most cases, a combination of these factors prevails and if the ensuing myocardial ischemia and anoxia are sufficiently severe or protracted, infarction or necrosis of the myocardium may develop. Myocardial necrosis may be either gross or microscopic. It is generally focal and disseminated, rather than massive and circumscribed, as in acute coronary occlusion. Characteristically, necrosis is localized in the subendocardial layer and the papillary muscles, the areas most susceptible to ischemia and anoxia. The endocardium and the pericardium usually are not affected.

The electrocardiogram of acute coronary insufficiency without acute occlusion is distinguished from the electrocardiogram of acute coronary occlusion by depression of the RS-T segment and inversion of the T-wave in one or more leads, without RS-T elevation or deep Q-waves. The changes, which often appear in all the standard and precordial leads, are generally reversible and

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transient, in contrast to the changes of acute coronary occlusion which are of long duration or permanent. The depression of the RS-T segment has been attributed to the subendocardial localization of the myocardial ischemia.[2]

Since the report of McGinn and White,[3] in 1935, on the electrocardiogram in pulmonary embolism, many authors have drawn attention to the so-called acute cor pulmonale pattern. This pattern is characterized by a deep S-1 and Q-3, depression of the RS-T interval in lead I and elevation of the RS-T interval in lead III, and inversion of T-3 or both T-2 and T-3. These changes have been attributed to acute right ventricular dilatation or strain resulting from the obstruction of the pulmonary artery. In our experience, as well as in that of other investigators,[4,5,6] this electrocardiographic pattern occurs in only a minority of cases of pulmonary embolism. In the majority of cases the electrocardiographic changes are less characteristic, consisting chiefly of RS-T depressions and T-wave inversions in one or more leads, without a deep S-1 or Q-3. Right ventricular strain cannot be considered the sole underlying cause of changes of this type since similar changes have been observed following acute coronary insufficiency precipitated by such causes as shock and hemorrhage.[2]

Horn, Dack and Friedberg[7] pointed out that acute coronary insufficiency following pulmonary embolism not infrequently resulted in ischemic changes in the left ventricular myocardium. These authors found either gross or microscopic infarction of the left ventricle, in the absence of acute coronary occlusion, in approximately one-fifth of 42 fatal cases which they examined. They believe that myocardial infarction was due to acute coronary insufficiency resulting from anoxemia and shock. Similar pathological observations were recently reported by Currens and Barnes,[8] who attributed the myocardial ischemia and infarction to increased right ventricular pressure and diminished coronary blood flow.

## *Material and Results*

This report is based upon a clinical and pathological study of forty fatal cases of massive pulmonary embolism (confirmed at autopsy) in which electrocardiograms were recorded following the embolism.

The electrocardiograms were classified in three groups: Group I consisted of 14 cases presenting the electrocardiographic pattern of acute cor pulmonale (Fig. 1). Group II consisted of 17 cases in which the electrocardiograms showed the changes of acute coronary insufficiency, namely, depression of the RS-T interval in two or more leads with or without inverted T-waves (Fig. 2).

Deep S or Q waves and distinct RS-T elevation were absent. In group III were placed nine cases of which 6 showed atypical QRS and T-wave changes, and 3 failed to show any recent changes. In the latter cases the electrocardiograms prior to the pulmonary embolism were very abnormal.

Atypical right bundle-branch block was observed in 3 cases of the cor pulmonale group (Group I). The conduction defect persisted until death in all 3 cases. In addition, various disturbances in cardiac rhythm were observed; nodal rhythm in 2 cases, supraventricular tachycardia in 2 cases, auricular fibrillation in 1 case, sino-auricular block in 1 case, and A-V block in 1 case.

A comparison was made of the influence of various predisposing and precipitating factors of coronary insufficiency in Groups I and II. These included age, antecedent cardiac or coronary disease, cardiac enlargement, right ventricular strain, and anatomic changes observed in the myocardium at autopsy. Electrocardiographic changes attributable to acute coronary insufficiency were

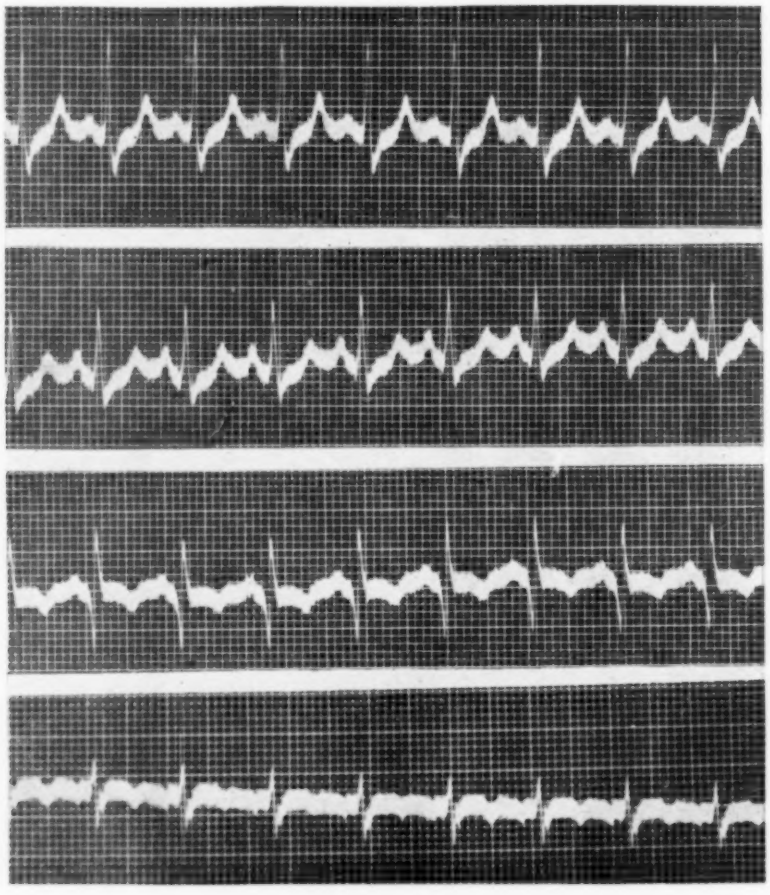

FIGURE 1

found for the most part in the older individuals and in those with clinical or anatomical evidence of antecedent coronary sclerosis and cardiac enlargement, more particularly in those whose previous electrocardiograms were very abnormal (Fig. 3). In other words, acute coronary insufficiency is more likely to follow pulmonary embolism in patients who are suffering from chronic coronary insufficiency than in individuals who have previously been free of cardiac abnormalities. On the other hand, the classical cor pulmonale pattern occurred most often in patients whose hearts were previously normal in size and free of coronary sclerosis.

Acute dilatation of the right ventricle without significant dilatation of the left ventricle was noted postmortem in almost one-half of the cases (45 per cent). In at least one-half of these cases the electrocardiographic changes were indicative of acute coronary insufficiency; the changes associated with acute cor pulmonale were not present.

Detailed anatomic study of the myocardium and the coronary arteries revealed acute myocardial necrosis or focal myocardial infarction of the left ventricle, in the absence of acute coronary occlusion, in 9 cases. The myocardial infarction was generally focal, disseminated, located in the subendocardial layer; in a number of instances it was localized in the papillary muscles of the left ventricle. Gross changes were visible in 3 cases (Fig. 4) and microscopic changes in 6 cases. These changes were like those found in postmortem examination of cases of acute coronary

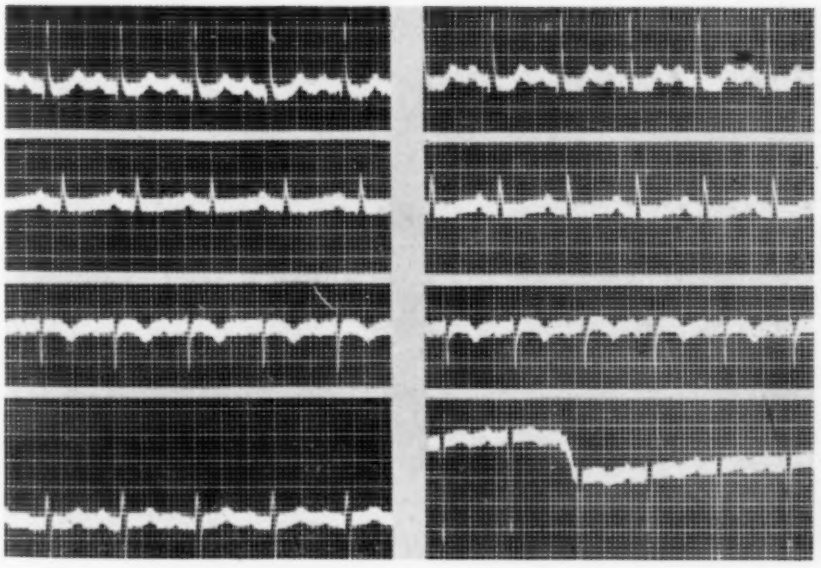

FIGURE 2

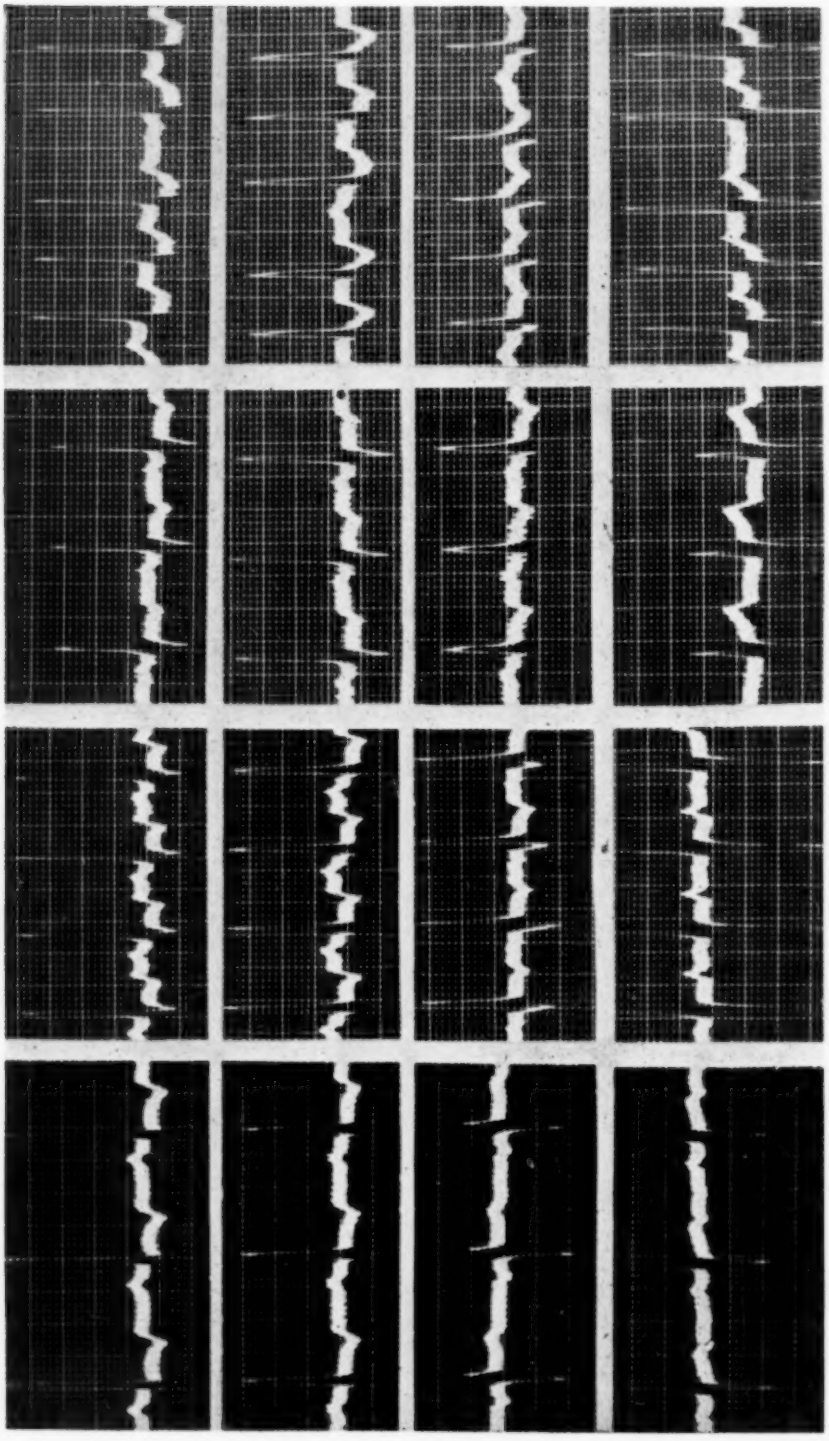

FIGURE 3

insufficiency precipitated by such factors as hemorrhage and shock.[2] In all nine cases, the left ventricle was involved, the anterior and posterior walls with equal frequency. Infarction of the right ventricle occurred in one case.

One of the nine cases with myocardial necrosis was in Group III, four were in Group I and four in Group II. Ischemic changes in the myocardium, then, occurred as often in patients with electrocardiographic signs of acute cor pulmonale as in those with acute coronary insufficiency. However, the myocardial changes in the coronary insufficiency group were more extensive and more severe than the changes that occurred in the cor pulmonale group. Gross myocardial infarction was observed only in the coronary insufficiency group (Group II).

Antecedent arteriosclerotic or hypertensive heart disease was present in all 9 cases of focal myocardial infarction. In four instances the coronary arteries were distinctly narrowed. In the remaining five cases, in which the coronary arteries were normal or only minimally narrowed, another factor predisposing to coronary insufficiency, such as hypertension and cardiac hypertrophy, was present. Five of the patients had sustained recurrent pulmonary emboli and had survived the initial spisode for two to three weeks (Figs. 2 and 3). The coronary insufficiency precitated by the recurrent pulmonary emboli was, therefore, sufficiently prolonged for anatomical myocardial changes to develop.[7] If death takes place soon after the occurrence of the embolism, myocardial infarction may not be demonstrable regardless of the severity of the myocardial ischemia.

*Discussion*

Clinical and pathological analyses of 40 fatal cases of massive pulmonary embolism lead to the conclusion that acute coronary

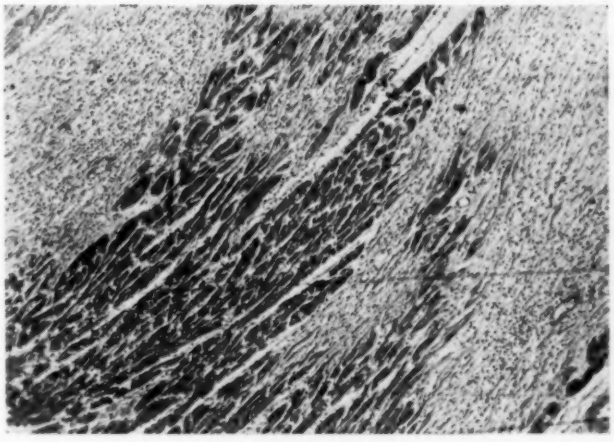

FIGURE 4

insufficiency plays as large a part as does acute cor pulmonale in the cardiac sequelae of pulmonary embolism. In our series the electrocardiographic signs characteristic of acute coronary insufficiency appeared more frequently than the changes associated with acute cor pulmonale. Moreover, coronary insufficiency not infrequently resulted in myocardial necrosis or infarction of the left ventricle.

Acute coronary insufficiency following pulmonary embolism may be caused by anoxemia, shock, right ventricular strain, and possibly reflex coronary vasoconstriction. Anoxemia, a marked clinical feature of pulmonary embolism, is manifested by dyspnea, tachypnea, and cyanosis. It is the result of diminished blood flow through the lungs and diminished arterial oxygen saturation. In spite of the fact that in experimental investigation[9,10] it has been found that a large cross section of the pulmonary artery must be mechanically obstructed before significant interference with the pulmonary circulation develops, it is well known that in human beings relatively small emboli may produce distinct pulmonary hypertension and circulatory effects. These effects have been attributed to widespread reflex vasoconstriction of the pulmonary arterioles mediated through the vagal nerve endings.[11] That profound vagal stimulation may occur was made manifest by the disturbances in cardiac rhythm that appeared in some of our patients and in animals following experimental pulmonary embolism.

Anoxemia and asphyxia may produce electrocardiographic changes indistinguishable from those produced by acute coronary insufficiency.[12,13] The underlying cause of the changes is anoxia of the myocardium, which, if intense or associated with insufficient coronary blood flow, may result in myocardial necrosis of the left and right ventricles.

Coronary insufficiency may be precipitated by shock, with its associated diminution of circulating blood volume, diminished cardiac output, diminished venous return to the heart and lowering of arterial blood pressure. Recent experiments[11,14,15] show that pulmonary embolism and increased pressure in the pulmonary artery are accompanied by immediate drop in systemic blood pressure and cardiac output. This drop was attributed to obstruction of venous return from the lungs to the heart[15] and to a reflex depressor mechanism between the lungs and the heart.[11,16,17] The hemodynamic effects of the shock and lowered systemic blood pressure will result in diminution of coronary blood flow.

When the coronary circulation is impaired by antecedent coronary sclerosis or cardiac hypertrophy and the myocardial ischemia secondary to the diminished coronary blood flow is severe or

prolonged, necrosis and infarction of the myocardium take place. The site of necrosis may be the left ventricle alone, or both the left and the right ventricles. Currens and Barnes[8] reported one case of isolated infarction of the right ventricle in pulmonary embolism, but this was not observed among our patients or in the series reported by Horn, Dack and Friedberg.[7] It may be concluded that the left ventricle is more susceptible than the right ventricle to the anoxic or ischemic effects of coronary insufficiency. The left ventricle is also the more susceptible in acute coronary occlusion, in which infarction of the right ventricle is rare except by direct extension from the left ventricle. The sites of predilection for focal infarction in the left ventricle in pulmonary embolism, as in other types of acute coronary insufficiency, are the subendocardial layer and the papillary muscles.

Reflex coronary vasoconstriction mediated through the vagus nerve[18] may be a factor in initiation of coronary insufficiency in pulmonary embolism. However, the validity of this reflex mechanism has been debated for many years, and recent experimental observations[15,19] support the opinion that this theory is ill founded. In any event, the hemodynamic effects of pulmonary embolism are so powerful that such a reflex mechanism need not be called into play.

Right ventricular dilatation and strain secondary to obstruction of the pulmonary artery is another agent responsible for precipitating coronary insufficiency in pulmonary embolism.[11] Right ventricular dilatation undoubtedly occurs,[20] since not infrequently it may be demonstrated during life,[4] and it was found at necropsy in almost one-half of the hearts in our series. However, it was not, in every instance, associated with electrocardiographic signs of cor pulmonale; in half of the cases in which isolated right ventricular dilatation was found postmortem, the electrocardiogram showed changes characteristic of acute coronary insufficiency. Furthermore, focal myocardial infarction of the left ventricle occurred as often in the presence of right ventricular strain as in its absence. It seems apparent that acute coronary insufficiency may be the predominant factor underlying electrocardiographic changes and myocardial involvement, even when right ventricular strain exists.

It has been observed experimentally[21] that increased right ventricular pressure may produce diminution in coronary blood flow, particularly in the right coronary artery, by increasing the resistance to blood flow within the coronary vessels. This may explain the resemblance of the classical pattern of acute cor pulmonale in the electrocardiogram to that of posterior wall infarction of the left ventricle, since the right coronary insufficiency may result

in greater ischemia of the posterior wall than the anterior. It may also result in greater ischemia of the right ventricle. Nevertheless, this is difficult to confirm postmortem, since the focal infarction is not localized to the posterior wall of the left ventricle but occurs with equal frequency anteriorly and posteriorly, and involves the right ventricle alone in only a small minority of cases.

## SUMMARY

A clinical and pathological study of 40 necropsied cases of massive pulmonary embolism indicates that acute coronary insufficiency plays as large a part as does cor pulmonale in the cardiac sequellae of embolism.

Electrocardiographic changes attributable to coronary insufficiency occurred more often than the classical cor pulmonale pattern. They were more common in older patients with antecedent coronary sclerosis and cardiac enlargement whereas the classical cor pulmonale pattern occurred most often in patients with normal coronary arteries and heart size.

Focal subendocardial infarction of the left ventricle, in the absence of acute coronary occlusion, was found postmortem in nine cases. This resulted from coronary insufficiency precipitated by the pulmonary embolism, in patients with antecedent coronary disease and cardiac hypertrophy who had sustained recurrent emboli.

The various precipitating factors of coronary insufficiency following pulmonary embolism were evaluated. These were anoxemia, shock, right ventricular strain, and reflex coronary vasoconstriction. Of these, shock associated with diminished cardiac output and coronary blood flow was considered the most important.

It can be concluded that acute coronary insufficiency may be the predominant factor underlying the electrocardiographic changes and myocardial involvement in pulmonary embolism, even when right ventricular strain exists.

## RESUMEN

Un estudio clínico y patológico de 40 casos autopsiados de embolia pulmonar masiva indica que la insuficiencia coronaria aguda desempeña un papel tan importante como el cor pulmonale en las secuelas cardíacas de embolias.

Alteraciones electrocardiográficas atribuíbles a la insuficiencia coronaria ocurrieron más a menudo que el patrón clásico del cor pulmonale. Esas alteraciones fueron más comunes en pacientes de edad más avanzada, con antecedentes de esclerosis coronaria e hipertrofia cardíaca; mientras que el patrón clásico del cor pul-

monale ocurrió más frecuentemente en pacientes con normales arterias coronarias y corazón de tamaño normal.

En el examen autópsico de nueve casos se encontraron infartos subendocardíacos focales del ventrículo izquierdo, sin oclusión coronaria aguda. Se debieron estos infartos a insuficiencia coronaria precipitada por la embolia pulmonar en pacientes con enfermedad coronaria e hipertrofia cardíaca antecedentes, que habían sufrido embolias repetidas.

Se avaluaron los varios factores que precipitan la insuficiencia cardíaca subsiguiente a la embolia pulmonar. Estos factores fueron: anoxemia, choque, tensión del ventrículo derecho y vasoconstricción coronaria refleja. Se creyó que el más importante de esos factores fue el choque asociado con la disminución de la cantidad de sangre que expela el corazón y disminución de la circulación coronaria.

Se puede concluir que la insuficiencia coronaria aguda puede ser el factor predominante de las alteraciones electrocardiográficas y de la lesión miocardíaca en la embolia pulmonar, aún cuando existe tensión del ventrículo derecho.

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# Discussion

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Brooklyn, New York

Dr. Master's and Dr. Dack's paper gives further additional evidence to reports appearing in the literature that the left ventricle is involved following pulmonary infarction, particularly in patients who have preexisting disease of the coronary arteries. The factor precipitating acute coronary insufficiency is undoubtedly the fall in blood pressure associated with shock that accompanies embolism to the lung. Currens and Barnes in 1943 reported 4 cases of myocardial infarction following pulmonary embolism involving the left ventricle wherein no significant obstruction was found in the coronary arteries to account for the acute enfarction and they suggested that probably shock contributed in a great measure to the decreased coronary blood flow. However they found only these 4 cases out of a series of 30 hearts examined.

In order to determine what our records at the Brooklyn Hospital showed regarding this problem, we examined the charts of 28 patients that succumbed to a pulmonary infarction. These records were not selected but taken in the order of their occurrence and represent patients from the various services of the hospital. Twenty of these individuals had preexisting heart disease. In 15, death was sudden and unexpected and due to massive embolization consequently no electrocardiographic records were available. Of these remaining 13, none showed the characteristic electrocardiographic changes described by McGuin and White, whereas in 2 of the charts there was some evidence that the myocardial infarct followed pulmonary embolization.

A female, age 54 years, who had hypertension was admitted to the Brooklyn Hospital for a gastro intestional survey because of a suspected neoplasm, as she complained of loss of weight and vague intestional symptoms.

On the 1st hospital day she was suddenly seized with pain in the chest, expectorated blood and went into shock with the development of signs of pulmonary edema. The leucocyte count was 20,400; 94 per cent polys and the electrocardiogram showed inversion of the T wave in leads I and IVF. Changes similar to these described in Dr. Master's paper. That she had a pulmonary infarction, there is no doubt, and it is very likely that the electrocardiographic changes represented acute coronary insufficiency involving the anterior wall of the left ventricle. She died 3 days following the acute episode and no autopsy was obtained.

A male, age 60, with hypertensive cardiovascular disease was admitted to the hospital following an acute myocardial infarction involving the anterior wall of the left ventricle. The electrocardiogram showed elevated R-T segments in lead I-II, and absent R and inverted T in lead IVF.

On the 10th day after admission he began to develop embolic manifestation in the lungs and following one of these episodes he went into shock. The electrocardiogram showed the development of deep Q2 and Q3 and elevated RT segments in leads II and III. It is interesting to speculate whether the pulmonary emboli were a causative factor in the development of the fresh infarct on the posterior surface of the left ventricle. No necropsy could be obtained.

Dr. Dack's study has given added emphasis to the occurrence of acute coronary insufficiency following pulmonary embolism and should make us more alert to detect evidence of this mechanism in the future. His studies may help us in explaining the series of events that developed in this patient.

A watchman, age 52, a diebetic of 4 years, was admitted to the medical wards of the Brooklyn hospital. He had no symptoms referable to his heart until one month before admission he complained of attacks of dyspnoea following climbing 2 flights of stairs. The night he entered the hospital he developed a severe attack of shortness of breath unassociated with pain in the chest. In the emergency ward he showed signs of shock and pulmonary edema. His heart was very rapid and the sounds were distant. The following day an x-ray of the lungs was reported as showing a zone of homogenous density in the right lower lung field. The area was triangular in outline with its base toward the chest wall and was highly suggestive of a pulmonary infarction. The electrocardiogram showed slight depression of the RT segments in leads 1, 2 and 3 and CF5 and was interpreted as a digitalis effect. Consultation with his local physician revealed the fact that he had been on Digitalis for the past 2 weeks.

Electrocardiogram taken 5 days later showed inversion of the T waves in lead 1, CF2, CF3, CF4 and CF5. These later changes indicated the possibility of anterior wall infarction. The tracing was again repeated 2 weeks later and it then showed a trend back toward the normal. There was less inversion in lead I and CF2 position and less inversion in CF3, 4 and 5. The x-ray was repeated at this time, and very scant

residual change were noted in the lower right lung field corresponding to the area of previously noted triangular density.

The series of events were a pulmonary infarction followed by electrocardiographic changes highly suggestive of acute coronary insufficiency.

From the work of Dr. Dack and from our own findings the electrocardiographic changes as described by McGuin and White following acute pulmonary infarction are not seen too frequently. However there is some evidence in our records to support Dr. Dack's contentions that the mechanism of acute coronary insufficiency may be a factor determining some of the cardiac effects of acute pulmonary embolism.

I am sure we all enjoyed Dr. Master's presentation and I am grateful to have had this opportunity of taking part in the discussion.

# The Diagnosis and Treatment of Acute Laryngotracheobronchitis*

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Rochester, Minnesota.

Acute laryngotracheobronchitis is a descriptive pathologic term for a disease of varied bacterial etiology. Despite its varied etiology the symptom complex generally fits into a definite pattern because the symptoms are produced by inflammation or obstruction of one or more of the breathing passages.

In past times it was generally assumed, and correctly so, that a child or an infant suffering from inflammation of the air passages or obstruction to breathing due to infection had diphtheria. That possibility should not be forgotten. Acute laryngotracheobronchitis, however, has occurred more commonly than has laryngeal diphtheria in recent years. It has been shown to have resulted from infection by hemolytic streptococci, Streptococcus viridans, Staphylococcus aureus, various pneumococci, Hemophilus influenzae and possibly on occasions by a virus.

The syndrome is seen in infants and children of all ages, but is more often a dangerous disease in children aged less than four years because of the relatively small size of the breathing passages. It is a disease most to be feared in those aged less than one year.

## *Clinical Picture*

The symptoms at onset of the disease are usually the same as those of spasmodic croup: a harsh brassy cough, and inspiratory stridor. These symptoms are generally due to subglottic edema, sometimes to supraglottic edema. Unless the vocal cords are involved the voice is not hoarse. Whatever the involvement of the region of the vocal cords, the classic signs of laryngeal obstruction are present: retraction above the suprasternal notch and clavicles, and of the epigastrium. Retraction of the intercostal spaces with inspiration is also often noted.

These symptoms at first may or may not be associated with fever. The absence of fever may correctly suggest that one is dealing with a case of spasmodic croup. However, my colleagues and I have seen several children whose dyspnea after an afebrile

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onset rapidly became very severe and who required tracheotomy within twelve to twenty-four hours after the onset. When dyspnea appears and increases in severity, fever and evidence of toxicity usually appear. If effective treatment is not instituted promptly, death may take place.

*Pathology*

As the name implies, acute laryngotracheobronchitis is an acute inflammatory process involving the larynx, the trachea and the bronchi. Often edema is the only pathologic change noted. Secretion soon appears, especially in the lower part of the trachea and in the bronchi, and causes considerable mechanical obstruction to breathing. The secretion may be thick, ropy, gummy and tenacious, and frequently causes bronchial obstruction and atelectasis (fig. 1). Inflammation and edema of the subglottic region are responsible for most of the symptoms of laryngeal obstruction

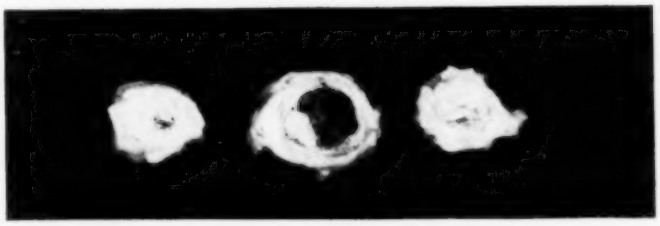

FIGURE 1: Purulent secretions obstructing a bronchus (hematoxylin and eosin x120).

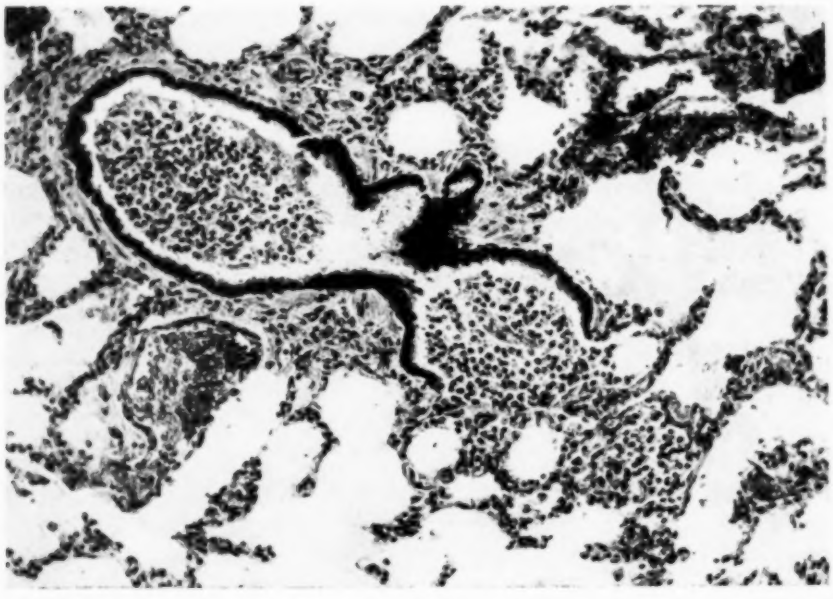

FIGURE 2: Cross sections of bronchi showing obstruction with thick secretions.

(fig. 2). Multiple regions of atelectasis may be present. The secretion may become so thick as to form a cast of the tracheobronchial tree.

### *Treatment*

The treatment of acute laryngotracheobronchitis is based on an understanding of the etiology and pathogenesis of the disease. Thick secretions must be thinned, an infectious agent combated and constant vigilance maintained for evidence of respiratory obstruction. In few other situations is constant intelligent nursing care so important.

Thick secretions are most readily thinned by thoroughly moistening the air which the patient breathes. A steam tent or steam room is usually the most convenient and effective means for supplying moist air. Two or three steam kettles are usually necessary to provide humidity. The water should literally drip from the top of the tent or from the ceiling. The more modern mechanical humidifiers provide moisture without at the same time making the room uncomfortably hot. Their use is to be preferred to that of a steam tent or steam room.

Oxygen should be well moistened if its use becomes necessary, since unmoistened oxygen exerts an undesirable drying effect on the secretion of the respiratory tract. Mechanical vaporizers[1,2] may be used in conjunction with an oxygen tent. Lacking one of these we recently have used two nebulizers instead. Distilled water was used as the fluid and an oxygen tank as the source of nebulizing force. In times past steam has been run into the oxygen tent by pipe or rubber hose. This method is cumbersome and unsatisfactory and may be dangerous. Moist cloths suspended in the partially filled ice compartment of an oxygen tent and moistened frequently provide sufficient moisture to maintain the humidity at 70 to 80 per cent. This, however, is not as high a humidity as is best to maintain.

It has been shown[3] that carbon dioxide is an effective agent for liquefying bronchial secretions. A 2.5 to 4 per cent mixture of this gas with 20 per cent oxygen in a tent has been suggested for treating pneumonia. My colleagues and I have not used it but we think it worth trying.

We often employ sodium or potassium iodide as an additional aid in liquefying tracheobronchial secretion. Five drops of a saturated solution of one of these salts may be given three times daily to a child of two years, and three drops to a child of one year. Davison[4] feels that the use of medicines is unnecessary if the inspired air is properly moistened and that the medicine may make the patient nauseated. We agree but we have seen children

who seemed to have been helped by the administration of one of the iodides. We have not tried to use any of the other so-called expectorant drugs.

It is essential that the patient's intake of fluid be maintained. If the patient cannot take fluids by mouth, they should be given by the intravenous or subcutaneous route. In infants at least 2 fluid ounces (60 cc.) per pound (0.5 kg.) per twenty-four hours should be given and in older children about 1 fluid ounce (30 cc.) per pound (0.5 kg.).

One or more small transfusions (100 to 150 cc.) of blood may be necessary as a supportive measure and to aid in combating the infection. The administration of concentrated blood plasma or blood serum has been suggested for its dehydrating effect on the laryngeal edema. We have not had any favorable results from its use in the few instances in which it has been tried. Concentrated human serum albumin might be tried.

Food may be given according to the child's desires. It is generally not desired during the acute phase except in liquid form.

It is very important that throat cultures be made. A suction apparatus such as was described several years ago[5] to collect material for typing of pneumococci is useful in obtaining material from infants for bacteriologic study. If tracheotomy is done, cultures of the tracheal secretions should be made.

Appropriate chemotherapy or antibiotic therapy varies from case to case depending on the etiologic bacterial agent. Penicillin is readily available at the present time and is effective when used to combat infections due to most strains of streptococci, staphylococci and pneumococci. It should be administered every two to three hours intramuscularly or by continuous intravenous infusion. Suggested dosage would be 200,000 units (divided into eight doses) daily for a child two years of age. If penicillin in a beeswax-oil medium is employed it is wise to use 200,000 to 300,000 units daily.

We prefer the use of procaine penicillin if a long-acting penicillin is to be used.

Large oral doses of penicillin can be given. In such acutely ill infants as those having acute laryngotracheobronchitis, one generally cannot depend on the oral route to provide adequate blood levels of the antibiotic agent.

Only rare strains of Hemophilus influenzae are killed by the use of penicillin. In fact one of the early uses of penicillin was to incorporate it in mediums on which Hemophilus influenzae was to be grown in order to kill off the other organisms which often overgrow it. Hemophilus influenzae infections respond well to the use of streptomycin. A total of 0.8 to 1.0 gm. daily, given intramuscularly in four to eight divided doses, is suggested. A daily

dose of 0.5 gm. will suffice in an infant less than one year of age.

The sulfonamide drugs still are valuable therapeutic aids. Hemophilus influenzae infections sometimes can be treated successfully with sulfadiazine, as more often can those due to streptococci, staphlococci and pneumococci. If sulfadiazine cannot be tolerated by mouth it may be given subcutaneously in the form of a 5 per cent solution of the sodium salt. The usual precautions regarding the use of the sulfonamide drugs must be observed.

Tracheotomy is indicated if the patient's dyspnea increases and if signs of laryngeal obstruction become more marked despite the use of moistening apparatus. Evidence of extreme restlessness, cyanosis and fatigue also is an indication for tracheotomy. In addition demonstration by auscultation that there is diminished entry of air into both lungs is further evidence that the patient's airway has been greatly reduced in caliber. Tracheotomy is preferred to intubation. Ideally it is done after a bronchoscopic examination and after crusts and secretions have been sucked out through the bronchoscope and while that instrument is still in place.

Great care must be used when performing a tracheotomy on an infant to see that the incision is made at a sufficient distance below the larynx so that the tracheotomy tube will not irritate the subglottal region. When this precaution is not observed undesirable subglottal scarring may result.

After tracheotomy the air entering the tracheotomy tube must be kept moist; this aids greatly in the prevention of crusts that tend to form in the trachea. The same measures for moistening the air may be used postoperatively as preoperatively. My colleagues and I have found, however, that the use of a nebulizer

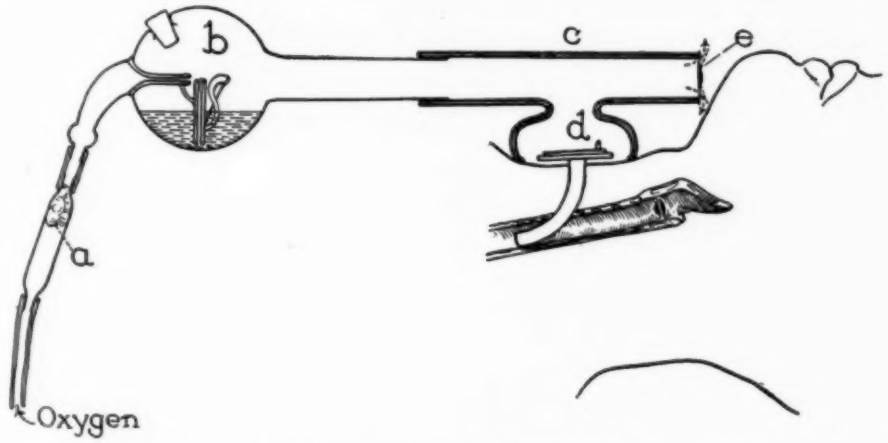


FIGURE 3: Nebulizer adapted to use for a tracheotomy tube. (From Albers, G. D.: "Use of a Nebulizer to Produce Oxygenated Vapor: Report of a Case of Acute Laryngotracheobronchitis," *Proc. Staff Meet., Mayo Clin.*, 18:511, Dec. 29, 1943).

as described by Albers[6] is most satisfactory (fig. 3). A nebulizer is attached to a rubber tube that carries oxygen. The distal end is applied to the tracheotomy opening by means of a flexible rubber hose. Iams[7] reported that a small rubber nipple with the teat end cut off is satisfactory for this purpose. In our experience this measure has been lifesaving. Subsequent bronchoscopic aspiration is often unnecessary; the tracheal secretions can be aspirated by a catheter because they are kept moist. Davison[8] urged that, when the suction catheter is used, it should not be inserted beyond the tracheotomy tube. Occasionally it is necessary to aspirate more deeply. When this is done very often the tracheal wall is irritated.

### *Contraindicated Drugs*

It is still felt by some physicians that the struggling dyspneic infant having laryngotracheobronchitis should be given a sedative drug. Such a practice is most unwise. While the infant is under the influence of sedatives the cough reflex is often suppressed, secretions accumulate in the breathing passages and the infant dies from suffocation. The sedative drugs obscure the signs and symptoms which might indicate a need for tracheotomy. Infants having laryngotracheobronchitis rarely struggle unless some obstruction to breathing is present. Therefore, the use of all opiates, barbiturates and other sedative or narcotic drugs is contraindicated.

Likewise it is not wise to administer atropine or its related compounds to an infant having acute laryngotracheobronchitis. Such drugs exert an undesirable drying effect on bronchial secretions.

Occasionally asthma and laryngotracheobronchitis have been confused. One might therefore be tempted to administer one of the so-called antihistaminic drugs. Because of their possible sedative effect and atropine-like action, their use is contraindicated in the treatment of acute laryngotracheobronchitis.

## SUMMARY

Acute laryngotracheobronchitis is a disease of varied bacterial origin. It is a very serious disease but more so when it occurs in children less than one year of age. Treatment consists in providing adequate moisture in the inspired air, sometimes the use of expectorant drugs, the administration of the proper chemotherapeutic or antibiotic agent, and careful nursing. Tracheotomy is sometimes necessary. Suggestions have been made regarding indications for its need, performance of the procedure and the after-treatment of the tracheotomized child. Sedative and antihistaminic drugs are contraindicated in the treatment of acute laryngotracheobronchitis.

## RESUMEN

La laringotráqueobronquitis aguda es una enfermedad de origen bactérico variado. Es una enfermedad muy grave, pero aún más cuando ocurre en niños menores de un año de edad. El tratamiento consiste de humedecer adecuadamente el aire inspirado, del uso, a veces, de drogas expectorantes, de la administración del agente quimioterapéutico o antibiótico apropiado y de una asistencia cuidadosa. A veces es necesaria la traqueotomía. Se han presentado sugestiones relativas a las indicaciones para este procedimiento, la manera de ejecutarlo y el cuidado subsiguiente del niño operado. Se contraindican los sedativos y las drogas antihistamínicas en el tratamiento de la laringotráqueobronquitis aguda.


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# Bronchography*

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Bronchography is an important diagnostic procedure and an essential part of a complete chest study when a diagnosis is not definitely proved by the usual history, physical examination, x-ray films and laboratory tests. Even when the diagnosis is known, information obtained from bronchograms may affect the type of therapy to be applied to certain patients.

It is the purpose of this paper to outline a simple technic which will make bronchography available to anyone who has fifteen minutes to spend with his patient and who has a fluoroscope and an x-ray machine available. Although the technic described was developed independently, it was later presented by Forrestier to the Southern Medical Association in 1937.

## *Technic*

Very little, if any, preparation of the patient is needed to make the routine or "screening" bronchograms filling both lower lobes, the right middle lobe, and the lower portion of each upper lobe. While it was once our practice to give nembutal an hour before, followed by codeine thirty minutes before the procedure, the majority of our patients now receive no medication. Only those patients who are unusually apprehensive, high strung, or uncooperative receive these preliminary drugs. It is unnecessary to withhold meals, although there is less saliva to interfere with a good filling of the bronchi if the procedure is carried out at least two or three hours after the patient has taken anything by mouth.

The equipment is simple, consisting of a 20 cc. syringe to which a 3½ inch piece of Carrel rubber tube is attached, a medicine glass with eyedropper, a piece of gauze, and a cup for warming the oil under running water. The anesthetic mixture consists of approximately 10 cc. of Pontocaine 0.5 per cent with 5 or 10 drops of adrenalin in it. We prefer the use of Iodochloral because its viscosity, when the oil is warmed, is just right for our purpose. Unless a large amount of the oil is swallowed by the patient, only 20 cc. is used.

Anesthetization is carried out with the patient sitting in a chair

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and leaning sharply to one side or the other while his head is supported by an assistant. The patient is asked to "stick out his tongue" (dentures having been removed) and the tongue is firmly grasped between the thumb and forefinger of the operator and withdrawn to prevent swallowing. The patient is then instructed to breathe through his mouth with "full, quiet breaths" while one or two dropperfuls of the anesthetic mixture are slowly instilled into the nostril which has the freer passage. The chief difficulty to be overcome is the tendency to swallow. An attack of coughing may ensue in which case the anesthetization is repeated until the operator is satisfied that the Pontocaine-adrenalin mixture has run well down the trachea without causing irritation. The patient is then tilted to the opposite side and the process is repeated through the same nostril. During the three of four minutes consumed by the process of anesthetization, the iodized oil and the syringe are warmed by running hot water over them.

Instillation of the iodized oil is performed in essentially the same manner and through the same nostril so that the oil flows over the membranes previously anesthetized. The rate of flow is varied with the type of breathing of the patient, but averages approximately 1.0 cc. for every two breaths.

The oil is directed into any lobe desired by placing the patient in an appropriate position *as it is instilled*. Satisfactory delineation of the bronchial tree is rarely obtained by positioning the patient *after* the filling has been completed. A little practice will make one adept at positioning the patient so that the desired bronchi will be filled. In general, a forty-five degree angle of direct lateral inclination will produce a filling of the *anterior* segments of the lower lobe, the middle lobe on the right or the lingula on the left, and those anterior branches in the upper lobes immediately above them. To obtain a filling of the superior segments of the lower lobes the patient must be tilted laterally and posteriorly. We usually start the instillation with a direct lateral inclination of the patient and gradually rotate him to a lateral and dorsal inclination.

It is possible to fill the upper lobes well by this method. When a filling of the upper segments and apex of the upper lobes is desired, the patient is seated and inclined almost horizontally in the direct lateral position, and is instructed to take *very full, deep breaths*. The warmed oil is carried into the upper bronchi in the same fashion as is inspired air, not by gravity, but by the negative pressure developed by the respiratory movement.

We do not advise an attempt to fill all the major bronchi with oil at one sitting. A very unpleasant sensation of suffocation can be developed and it is not impossible that certain patients with

impaired respiratory function and children could actually be asphyxiated. Rather, we prefer to make our routine bronchograms in such a fashion that the upper segments of each upper lobe are free of oil, to be filled at a different sitting if indicated. Or in case our preliminary examination indicates pathology in the upper lobes, we will perhaps fill all of one lung at one sitting, leaving the contralateral lung free of oil at the time.

For routine purposes the right lung is filled first in front of the fluoroscope and when the desired bronchi have been delineated a *right lateral* x-ray film is made. This will clearly outline the bronchi of the various segments without interference from shadows in the contralateral lung. It is also possible to be much more certain of the identification of various branch bronchi in the true lateral film than in the diagonal view where a portion of the bronchial tree may be partially obscured by the heart shadow.

Then in a similar fashion the left bronchi are outlined and a postero-anterior film is made. In some cases a left anterior oblique film may be useful.

Stereoscopic x-ray films of iodized oil instillations are frequently used in place of the foregoing group of films. In them it is still easier to identify the bronchus of each lobule of every lobe filled.

There are a few precautions which make the difference between good and useless bronchograms: (1) It is important that the x-ray films be made *immediately* after the instillation of oil, before there has been an opportunity for the bronchial outlines to be clouded by deposits of oil in the alveolar spaces. (2) Iodochlorol flows best and reaches its ideal viscosity when *warmed* to a little above body temperature. It is also least likely to cause the patient to cough when properly warmed. (3) The use of too much oil will obscure the structures which are being examined; namely, the bronchi. It is usually sufficient to use 10 cc. on each side.

Definition is improved by the use of a grid, either of the Bucky type or one which is stationary and can simply be hung on the cassette holder between the patient and the film. In any case the penetration must be adequate to outline the oil in the bronchi *through* the heart shadow and *through* the shadow of the diaphragm and liver. This can readily be achieved with ordinary x-ray equipment and without materially increasing the time of exposure by using its maximum capacity and, in many instances, shortening the distance from the usual 72 inches to 42 inches. The intensity of the exposure varies inversedly with the square of the distance so that this change will result in approximately three times the penetration achieved by the same factors at six feet.

Immediately following the instillation of oil and the making

of x-ray films, every patient is given a postural drainage. He is further instructed to take postural drainage *three* or *four times more* before retiring. This is performed by assuming an angle of forty-five degrees with the floor, lying over a bed or examining table, and coughing forcefully for two or three minutes to evacuate all the oil possible. The majority of patients will be entirely free of oil by the following day. This is an important maneuver in the prevention of lipoid pneumonia, atelectasis or other complications.

This is not the only effective technic for making bronchograms, and for certain special indications and in small children other technics must be followed. Excellent bronchograms in children can be made through the bronchoscope if the bronchoscopy is carried out on the x-ray table, or if a portable x-ray machine is placed in position in the bronchoscopic room during the introduction of the oil. To obtain satisfactory bronchograms it is important that the films be made immediately upon the introduction of the oil into each lung individually. All oil possible is withdrawn from each lung through the suction apparatus and the child is given continuous postural drainage with the foot of the bed elevated until the chest is clear.

Aside from an occasional patient whose salivary glands become swollen and painful as a result of sensitivity to iodine we have had no untoward results in well over two thousand sets of bronchograms made by the technic described here.

### *Indications*

When bronchography is easily and readily available, its uses are found to be more numerous. Besides the traditional use of bronchography for the identification of bronchiectasis and a determination of its distribution and extent for surgical purposes, it is an invaluable aid in tracing down the cause of hemoptysis of unexplained origin. A persistently blocked bronchus as observed on more than one bronchogram may well mean a previously unsuspected foreign body, an adenoma of the bronchus, a carcinoma or other lesion which fails to cast a shadow on the normal roentgenogram. It is also amazing to find at times how much bronchiectasis may exist, causing hemoptysis, when the patient fails to give a history of persistent cough and sputum.

Some patients suspected of having bronchiectasis from the history alone are found to have generally attenuated bronchi. The associated cough and occasional wheezing are found to be due to an allergy—asthma without marked wheezing.

In the distal bronchi which are not readily visualized with a bronchoscope, one may identify the bronchus in which a foreign

body is situated by means of bronchograms, thereby facilitating its removal bronchoscopically.

In patients in whom it is difficult to differentiate areas of atelectasis from localized pockets of fluid or extrapulmonary tumor masses, bronchograms provide the indisputable differential diagnosis.

A generalized density about the base of the thorax makes it impossible in some cases to tell whether or not pathology is in the lung or is located below the diaphragm and bronchograms again will provide valuable information.

In tuberculosis the use of iodized oil has been attended by no untoward complications. We no longer hesitate to instill iodized oil in the patient who has had tuberculous disease and who has unexpected or unexplained positive sputum. Tuberculous bronchiectasis and tuberculous tracheo-bronchitis have both been demonstrated in many instances as the cause of this phenomenon.

There are other special indications for bronchography. If the examination can be quickly and easily performed without any particular distress to the patient, the chest diagnostician will find it one of his most useful and frequently used tools.

## SUMMARY

We have outlined a technic for bronchography which is simple, easy, and which may be carried out as an office procedure without elaborate preparation or distress to the patient. It is effective for routine purposes and in the majority of cases. A few of the indications for bronchography are outlined.

## RESUMEN

Hemos bosquejado una técnica para la broncografía que es sencilla, fácil y que puede llevarse a cabo como procedimiento de oficina sin preparativos esmerados o dolor al paciente. Es eficaz para fines rutinarios en la mayoría de los casos. Se bosquejan algunas de las indicaciones para la broncografía.

# Cavity Closure Following the Discontinuation of Ineffective Pneumothorax*

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Judging by the paucity of reports, rapid and unexpected closure of tuberculous cavities after the discontinuation of an ineffective pneumothorax is a rare occurrence. In 1938 Steele, et al,[1] reported 12 cases in which cavity closure occurred unexpectedly after the discontinuation of an ineffective pneumothorax. Since it is the only such report in the literature an additional report of 11 cases seems worthwhile.

The first 5 cases were observed in a period of forty-four months in a large sanatorium of 665 beds. Cases 6 to 11, inclusive, were seen in a recent period of eighteen months in a small sanatorium (C.C.S.) with an average of 70 adult patients.

In most of our patients, phrenic paralysis was substituted immediately after the pneumothorax was discontinued. In recent months phrenic paralysis has been virtually routine, except in cases with specific contraindications. Ten of our 11 cases had phrenic paralysis. Five of the 10 also had pneumoperitoneum. Pneumoperitoneum has not been routine because in many instances the diaphragm becomes sufficiently elevated after phrenic crush to obviate the need for pneumoperitoneum. If the diaphragm does not rise to a high level within a period of two or three weeks pneumoperitoneum is induced for the purpose of further relaxing the lung.

In selected patients pneumoperitoneum is an effective method in the treatment of tuberculosis. Its usefulness has not been generally appreciated. Although this is not intended as a report on the merits of pneumoperitoneum, the role of pneumoperitoneum is well illustrated by the fact that in the last eighteen months during which this form of therapy has been used, five such cases have been seen, whereas in the previous forty-four months there were only five cases.

Probably, every phthisiologist has observed patients with ineffective pneumothoraces whose lungs after reexpansion showed un-

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expected improvement characterized by resorption of exudate, decrease in size (or closure) of cavities and contraction of the diseased portion of the lung. Such rapid improvement is rarely noted in patients treated solely by bed rest. It seems paradoxic that so much improvement should follow the discontinuation of an anatomically poor and clinically ineffective short-term pneumothorax.

An anatomically perfect pneumothorax produces concentric collapse, relaxation and diminution in motion of the diseased portion of the lung. Some degree of relaxation also occurs in many anatomically poor but clinically effective pneumothoraces. This relaxation and contraction assists in the resolution of foci of infiltration. These favorable changes are the basic aims of collapse therapy.

It is not the purpose of this paper to discuss the various hypotheses of cavity closure. Suffice it is to say that cavity closure depends on such factors as atelectasis, compensatory emphysema, fibrotic contraction of cavity wall and occlusion of bronchocavitary communication. It is believed that no single factor accounts for cavity closure but that several play variable roles.

Steele et al,[1] offered no explanation for cavity closure in their patients. The only explanation I can advance is the already known mechanisms of cavity closure which are aided by the relaxation which begins with the ineffective pneumothorax and continues with phrenic paralysis and pneumoperitoneum. It is customary in collapse therapy to date a patient's improvement from the day an effective collapse is achieved by one method or another. In view of the benefit derived from many so-called ineffectual pneumothoraces, one must admit that improvement begins and coincides with the period of so-called ineffectual pneumothorax. Conversion of sputum occurs in some so-called anatomically poor pneumothoraces. In other anatomically poor pneumothoraces sufficient relaxation occurs to permit the diseased portion of the lung to contract and occupy a smaller space. This contraction has been repeatedly observed in right upper lobe disease where the lobar fissure before pneumothorax is horizontal but after reexpansion is found to follow an ascending plane in the lateral direction, indicating contraction of the entire lobe. Further complete contraction of a badly diseased right upper lobe into the apex and against the mediastinum has been seen occasionally after discontinuation of an ineffective pneumothorax. This rapid decrease in volume of an entire lobe is attributed to the beneficial effect of the short term pneumothorax.

Even though a lung may be broadly adherent in the upper portion, unless the base is solidly adherent also, some relaxation

TABLE 1 — ANALYSIS OF ELEVEN CASES

| Case | Age and Sex | Date of Admission to Sanatorium | Duration of Pneumothorax | Date of Phrenic | Time from Phrenic Crush to Cavity Closure | Pneumoperitoneum | Status on Discharge from Sanatorium | Status on Last Observation | Comment |
|---|---|---|---|---|---|---|---|---|---|
| Case 1 O.F. | 24-F | 4/16/43 | 2 mos. | 6/30/43 | 9 mos. | No | 12/5/44 Arrested | Not Traced | Closed 4 cm. left apical cavity. |
| Case 2 R.A. | 32-F | 7/22/43 | 6 wks. | 9/24/43 | 11 wks. | No | 9/4/44 Arrested | Not Traced | Closed 2.5 cm. right apical cavity. |
| Case 3 O.C. | 21-F | 10/26/42 | 5 wks. | 11/14/42 | 9 mos. | No | 4/24/44 Arrested | Not Traced | Remarkable result. Closed 7.5 cm. right upper lobe cavity. |
| Case 4 D.W. | 50-F | 8/18/42 | 9 wks. | 9/27/43 | 12 wks. | No | 2/11/44 Quiescent | Not Traced | Closed 2.5 cm. right upper lobe cavity. |
| Case 5 G.I. | 27-F | 7/ 8/43 | 8 wks. | Not done | 7 mos.* | No | 9/5/44 Arrested | Not Traced | Closed 3 cm. right upper lobe cavity. |
| Case 6 R.D. | 18-F | 7/23/45 | 11 wks. | 11/ 1/45 | 9 wks. | No | 2/1/46 Quiescent | Arrested 3/15/47 | Closed 3 cm. right apical cavity. |
| Case 7 W.C. | 36-M | 5/23/45 | 10 wks. | 5/24/45 | 8½ mos. | Yes | 11/10/46 App. Arres. | Arrested 3/15/47 | Closed 4 cm. left apical cavity. |
| Case 8 D.H. | 26-F | 5/27/46 | 2 wks. | 6/17/46 | 5 mos. | Yes | Still in Sanatorium | Sputum Neg. Doing well | Has diabetes. Closed 4 cm. left apical cavity. |
| Case 9 B.C. | 16-F | 4/ 9/46 | 2 wks. | 7/ 6/46 | 6 mos. | Yes | Still in Sanatorium | Sputum Neg. Doing well | Closed 4 cm. right apical cavity. |
| Case 10 C.D. | 24-M | 10/18/45 | 8 wks. | 12/27/45 | 10 mos. | Yes | Still in Sanatorium | Sputum Neg. Doing well | Closed 5.5 cm. right upper lobe cavity. |
| Case 11 A.M. | 31-M | 6/ 4/46 | 3 wks. | 7/ 3/46 | 5½ mos. | Yes | 3/18/47 App. Arres. | Apparently Arrested | Closed 4.5 cm. right apical cavity. |

*Seven months from induction of pneumothorax.

occurs in an apico-basal direction. When the patient is in bed, the pull of gravity is overcome in an apico-basal direction allowing the lung to retract upward. Pneumothorax more or less releases the lung from the pull of the diaphragm. Phrenic paralysis and pneumoperitoneum further the upward retraction of the lung.

Cavity closure requiring several months may not be considered rapid by some but the closure of cavities in Cases 2 and 4 in a few weeks seems significant, particularly in Case 4. This patient had a persistent 2.5 cm. cavity for several months but refused pneumothorax. She finally agreed to the induction of a right pneumothorax but the collapse was ineffective because of dense pleural adhesions. Twelve weeks elapsed from the date of induction to complete reexpansion of the lung, following which the cavity was found closed and remained so. This case illustrates more than any other the beneficial effect of a short term ineffective pneumothorax.

No case with cavity smaller than 2.5 cm. in diameter is included in this group. The shortest cavity closure time seen was in Case 2 in which a 2.5 cm. cavity closed in eleven weeks. The largest cavity (Case 3) measured 7.5 cm. in diameter and closed in nine months. Smaller cavities in other cases required a longer time to close. In general, there was no correlation between the size of the cavity and the length of time required for closure. Most of the cavities were of recent origin; the walls were not thick and no doubt belong to Pinner's Type 2. None were Pinner's Type 3. In all cases the sputum remained positive two to three months after the cavity closed. In eight of the patients the cavity was located in the right upper lobe while in three it was in the left upper lobe.

The general condition on admission of the 11 cases listed in Table 1 was better than average. An unusual feature which is not brought out in the table is that the contralateral, or so-called good lung, in all except 3 cases, showed no roentgenographic evidence of disease. In these 3 cases there was minimal exudative disease which regressed satisfactorily. The clinical and radiographic progress of these patients has been excellent.

Because of the frequency with which empyema complicates ineffective pneumothorax, early discontinuation has been the rule in our work. Eleven weeks was the longest pneumothorax trial period in these cases and there were no complications.

## SUMMARY

Eleven cases illustrating the closure of tuberculous cavities following the discontinuation of ineffective pneumothorax have been tabulated and analyzed.

Phrenic paralysis was substituted for the pneumothorax in 10 of the 11 cases. Pheumbperitoneum supplemented phrenic paralysis in 5 cases. Phrenic paralysis and pneumoperitoneum were effective adjuncts in the treatment of these cases.

Certain principles of collapse therapy and their influence on cavity closure in these cases are discussed.

Cases 6 to 11 inclusive have been followed since this paper was submitted for publication. At this time all have arrested disease.

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I wish to thank Dr. G. E. Gwinn, Superintendent, Pinecrest Sanatorium, Beckley, West Virginia, for the privilege of reporting Cases 1 to 5 inclusive, which I observed while a member of his staff.

## RESUMEN

Se han presentado en forma de tabla y se han analizado 11 casos que ejemplifican el cierre de cavernas tuberculosas subsiguiente a la discontinuación de neumotóraces ineficaces.

En 10 de los 11 casos se reemplazó al neumotórax con la parálisis del frénico. En 5 casos el neumoperitoneo reforzó la parálisis del frénico. La parálisis del frénico y el neumoperitoneo fueron adjuntos eficaces en el tratamiento de estos casos.

Se discuten ciertos principios de la colapsoterapia y la influencia que ejercieron sobre el cierre de las cavernas en estos casos.

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# Pleural Fibrin Body Simulating Cardiac Aneurysm: Report of a Case

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In recent years, roentgenologic study has come to be recognized as the most accurate method in the diagnosis of left ventricular aneurysm.[1-6] Generally there is an abnormal contour of the cardiac shadow with a localized bulge somewhere along the ventricular border. A paradoxical pulsation at the site of the aneurysm may be helpful but it is not always present. The apex of the heart is said to be the most frequent location for aneurysms but this type may be most difficult to detect due to the fact that the abnormal ventricular bulge is partially hidden by the shadow of the left diaphragm. Films taken in deep inspiration may be of some help. Emphasis has justly been placed on the importance of fluoroscopy in the proper evaluation of an abnormal cardiac silhouette. This is true not only in the identification of various types of heart lesions but also in their differentiation from noncardiac disease.

The case reported below showed a localized shadow in the region of the cardiac apex which resembled a ventricular aneurysm. However, fluoroscopy showed that the suspicious density was caused by a large fibrin body which had lodged at the bottom of the left pleural space so that it seemed to be continuous with the cardiac shadow.

## CASE REPORT

R.T.A., a 37-year-old white male was transferred from another institution for treatment of his tuberculosis. He had worked as a jeweler for 17 years and had always been in good health until one year ago when he noted the onset of cough, slight dyspnea, and easy fatigue while he was with the U. S. Army in Europe. In July, 1946 he had a ten-ounce hemoptysis and roentgenograms of the chest at a Veterans Administration hospital showed a tuberculous process in the left upper lobe with a 3-centimeter central area of increased translucency which was interpreted as a cavity. Pneumothorax was instituted in September, 1946 although no definite tubercle bacilli had been isolated from the sputum. He was transferred to the Veterans Administration Hospital, Oteen, North Carolina on September 24, 1946.

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*From the Department of Medicine and Surgery, Veterans Administration, Oteen, North Carolina; published with permission of the Chief Medical Officer, Department of Medicine and Surgery, Veterans Administration, who assumes no responsibility for the opinions expressed or conclusions drawn by the author.

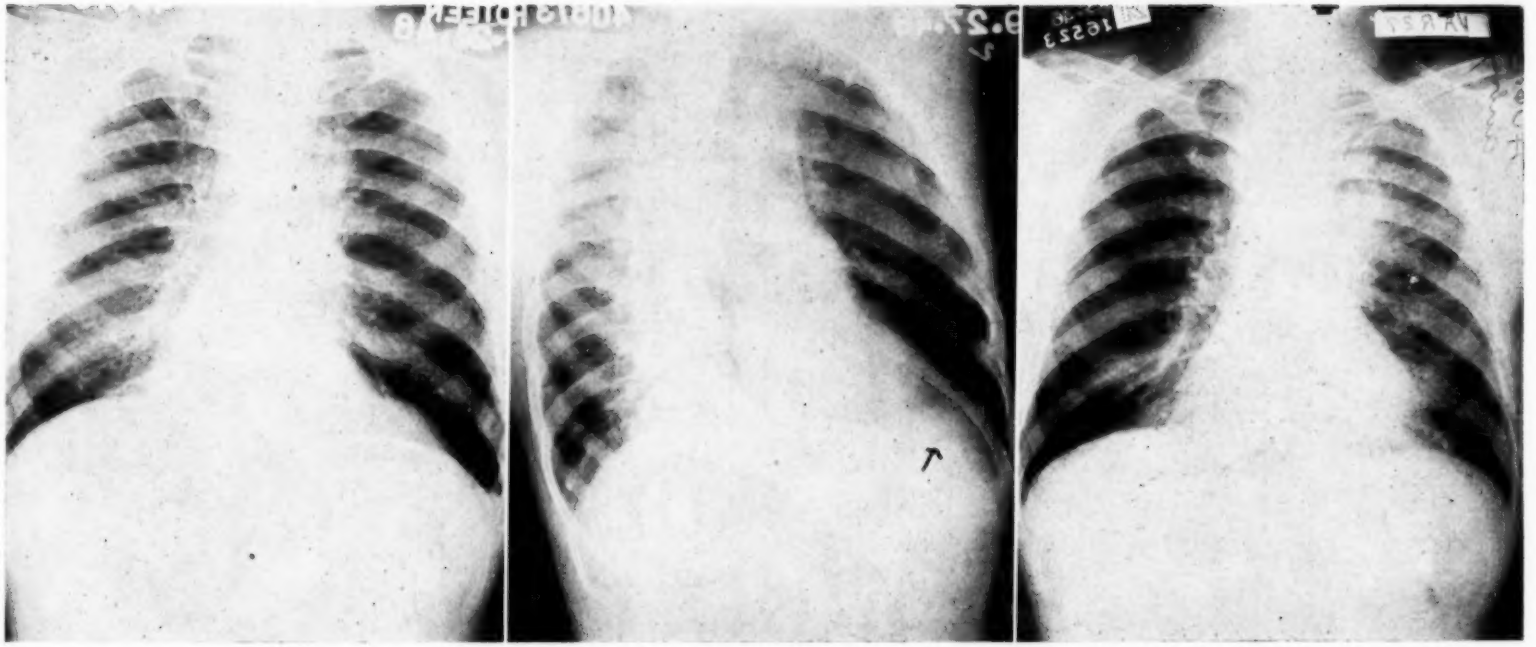

FIGURE 1 FIGURE 2 FIGURE 3

*Figure 1:* Admission film of September 24, 1946 demonstrating abnormal shadow in the region of the cardiac apex. Arrow indicates the small incisura separating the oval density from the normal left cardiac border.—*Figure 2:* Right anterior oblique view of chest showing large egg-shaped fibrin body in left pleural space (indicated by arrows).—*Figure 3:* Pre-pneumothorax chest film of patient, dated Aug. 23, 1946, showing cardiac shadow of normal size and shape.

His admission chest film (Fig. 1) at this hospital showed a pneumothorax on the left side with about 25 per cent collapse of the left lung and a minimal amount of fluid in the left costophrenic sulcus. Dense infiltration was present in the medial portion of the left upper lobe just above the hilar vessels. No definite cavities were seen. Right lung was clear.

The cardiac silhouette was abnormal with a convex density adjacent to the cardiac apex and separated from it only by a small incisura. The appearance was suggestive of an aneurysm of the apex of the heart. However, this patient gave no history of any preceding coronary occlusion or any definite symptoms of heart disease. Auscultation over the precordium was essentially negative and the blood pressure was 120/82 Electrocardiogram was normal.

The patient was fluoroscoped in various positions and it was found that the abnormal shadow adjacent to the cardiac apex was caused by a large egg-shaped fibrin body resting in the antero-medial portion of the left pleural sac at the level of the cardiac apex. This is well demonstrated in the right anterior oblique view of the chest (Fig. 2).

The chest film done on this patient before induction of pneumothorax was later secured from the institution where he had originally been hospitalized. It showed a heart shadow of normal configuration. The abnormal density at the apex had apparently developed only after the induction of the pneumothorax (Fig. 3).

Pleural fibrin bodies are known to develop in a certain percentage of pneumothorax cases. Some authorities regard injury to an intercostal vessel or tearing of an adhesion band as a prerequisite to the formation of fibrin bodies. However, there are cases in which the fibrin body seems to develop within a pleural exudate with no definite history of a traumatic episode. In such cases there is usually present an appreciable amount of pleural fluid before the fibrin body makes its appearance. Usually a fibrin body will move freely in the pleural exudate when the patient is postured and will thus be easily identified on fluoroscopy. Once it becomes adherent, however, either to the diaphragm or to the wall of the pleural cavity, diagnosis becomes much more difficult.

## SUMMARY

A case is reported in which a large fibrin body lodged in the left pleural space in such a position as to simulate an aneurysm of the apex of the heart on the postero-anterior film of the chest. Fluoroscopy and oblique projection, together with comparison of pre-pneumothorax roentgenogram revealed the true nature of the condition.

## RESUMEN

Se informa sobre un caso en el que un cuerpo grande de fibrina paró en el espacio pleural izquierdo en tal posición que, en la película postero-anterior del pecho, aparentó ser un aneurisma

del vértice del corazón. El examen radioscópico y la proyección oblícua, comparados con una radiografía tomada antes de comenzarse el neumotórax, revelaron la verdadera naturaleza del caso.

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# A New Mirror-Cannula for Laryngo-Tracheo-Bronchial Anesthesia, Medication, or Instillation of Opaque Oils

A. ALBERT CARABELLI, M.D., F.A.C.P., F.C.C.P.*
Trenton, New Jersey

Frequently there comes to the laryngologist, bronchoscopist, and pulmonologist a desire to possess three hands. This is especially true during the process of anesthetizing the larynx, trachea, or bronchi. The usual procedure is to hold the mirror in one hand, the cannula in the other, using the patient or an assistant to make traction on the tongue. This means a total of three hands about the mouth orifice. Traction of the tongue by an assistant or the patient has a considerable personal variable which ranges from too much traction resulting in pain and gagging to too little resulting in almost complete block of the line of vision for the operator by the elevation of the dorsum of the tongue. If the patient holds the tongue, there is a constant tendency to retract it into the mouth even with the most cooperative patient. Manipulative dexterity on the part of the operator has in many cases developed skills that make anesthesia of the larynx and trachea relatively simple procedures. On the other hand some operators even after many years find this procedure cumbersome and occasionally awkward, especially with certain types of patients. Occasionally the tension during this procedure has been such that an unsuccessful anesthesia has resulted or the patient has become unwilling to continue with the ordeal.

In view of the personal difficulties with the old procedure which seem inherent in the method, the author has attempted to simplify the procedure of laryngo-tracheo-bronchial anesthesia by eliminating the use of the third hand and by making other refinements which result in a rather smooth technique without at any time obstructing the line of vision. This was done by devising a simple mirror-cannula which is described below. Proficiency in the use of this cannula comes rapidly; and at present what was once an ordeal with certain patients has become a matter of routine. With the use of the mirror-cannula complete control is had at all times on the degree of traction of the tongue and the solution is instilled accurately and economically where intended.

Prior to developing the mirror-cannula consideration was given

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to the ideal requirements for successful and simple anesthesia of the larynx, trachea, and bronchi. It was decided that were a single instrument designed that would permit conformance with the following requirements, an ideal instrument for this procedure could be evolved:

1) A procedure that could be performed by the operator alone without the assistance of the patient or nurse.
2) Lack of bulk to the instrument for easy introduction into the mouth without eliciting undesirable reflexes.
3) Design of such an instrument to closely follow the natural oro-pharyngeal curve without touching too many structures in the mouth, pharynx or larynx.
4) Incorporation into the instrument of a means of holding back the flaccid uvula so that it did not fall unto the mirror and obstruct the line of vision.
5) An instrument that permits an unobstructed view at all times of the posterior portion of the epiglottis and of the vocal cords.
6) An instrument that permits complete control of the solution to be instilled under constant visual guidance.

After much experimentation, mostly by the trial and error method, a mirror-cannula was developed that permitted the above desiderata. Such a cannula is illustrated in Figure 1. The basic parts consist of a stock laryngeal mirror (No. 4) and an Abraham laryngeal cannula of malleable metal. The shortened shank of the mirror is simply soldered to the back of the cannula after molding by hand to the required shape. Multiple clinical trials finally evolved a mirror-cannula with specifications which were considered generally satisfactory without further modification for anesthesia of the larynx, trachea, or bronchi. The following are the specifications for the mirror-cannula:

1) Size of mirror: Number 4 laryngeal mirror, boilable.
2) Length of shank of mirror: 4.5 cm. from edge of attachment to mirror.

FIGURE 1

3) Angle of mirror to the horizontal line: 45 degrees.
4) Angle of hub of cannula to the horizontal line: 30 degrees.
5) Angle of the descending portion of the cannula to the horizontal line: 110 degrees.
6) Distance from the center of the mirror to the curve of the cannula: 0.5 cm.
7) Distance from the center of the mirror to the rounded tip of the cannula: 3.5 cm.

The angle of the mirror to the shank is changed to conform with specification number 3, above, as the original angle with which the mirror comes is not the same. Changing of this angle is made close to the edge of the attachment of the shank to the mirror with the aid of a narrow-nose wire-bending pliers so as not to crack the glass.

Experimentation with sizes 1 to 6 of the laryngeal mirror, but using the same basic specifications proved that the ideal size of mirror was a number 4. The smaller sizes permitted the uvula to occlude vision when it slipped over the anterior portion of the mirror. The larger sizes were found too bulky for introduction and gave an unnecessarily large view of the vocal cords and the surrounding structures.

Variations of the length of the tip, keeping the other specifications constant also revealed enough difficulties to warrant standardization to the 3.5 cm. length of the tip. Shorter distances may be used but have the disadvantage that the tip cannot be placed over the posterior portion of the epiglottis so as to direct the solution between the vocal cords. Generally it was found that with the shorter tips the solution trickled down the anterior portion of the epiglottis and traveled into the pyriform sinuses eliciting a swallowing reflex which made for loss of the solution into the esophagus and failure to anesthetize the larynx. Another disadvantage was that the epiglottis could not be engaged by the tip of the cannula and thus secure its position just above the vocal cords. Longer distances than 3.5 cm. also had several disadvantages. One was that the tip of the cannula became captured between the vocal cords before they could be anesthetized and caused spasm, cough, and gagging. Another disadvantage was that introduction of the longer tip into the natural oro-pharyngeal curve was cumbersome and evoked gagging and salivation.

The 3.5 cm. length of the tip was found to be ideal since it permitted easy introduction into the mouth along the natural oropharyngeal curve with only two points of contact: the uvula and the upper, posterior portion of the epiglottis. Since these structures are usually well anesthetized by the preliminary spraying with an atomizer, no undesirable reflexes are elicited when they are touched by the cannula. The posterior portion of the mirror is used to

gently push back the flaccid uvula thus keeping it out of the field of vision and the tip is made to rest gently against the upper posterior portion of the epiglottis at such an angle that the vocal cords are well visualized, permitting the solution to be dropped directly upon them or between them at will without setting up the severe reflexes noted when the tip touched the cords or lies between them.

Attached to the mirror-cannula is a standard Luer-lock syringe which contains the solution to be used. The angulation of the hub is such that vision is not impeded at any time by the bulk of the syringe and has the added advantage that the hand can be kept at a low position and is not in the line of vision.

A feature worthy of mention of this mirror-cannula is that since the tip is also malleable its angle with the horizontal may be changed to suit the occasional patient who has the epiglottis at an angle varying from the normal. By manual angulation of the tip, introduction behind the epiglottis can be accomplished with almost any anatomical variation of the epiglottis. This contingency is apparently rather rare and the degree angle of the tip was changed only once in a series of 180 anesthetizations.

Use of the mirror-cannula, of course, presupposes preliminary anesthetization of the oro-pharyngeal and superficial laryngeal structures with a spray anesthesia from an atomizer as is done with the usual method. Fogging of the mirror is also controlled by heating in the flame of a spirit lamp or hot water.

## SUMMARY

1) A simple indirect vision mirror-cannula is described which simplifies laryngo-tracheo-bronchial anesthesia, and instillation of medication or opaque oils.

2) The mirror-cannula is simply constructed of standard parts and can be easily and expeditiously assembled.

3) Use of the mirror-cannula obviates the use of the patient or an assistant to make traction on the tongue.

4) The optimum specifications for such a mirror-cannula are described.

## RESUMEN

1) Se describe un sencillo espejo-cánula de visión indirecta que simplifica la anestesia laringo-tráqueo-bronquial y la instilación de medicinas o aceites opacos.

2) Se construye sencillamente este espejo-cánula, usando partes regulares que se pueden armar fácilmente y con prontitud.

3) Con el empleo del espejo-cánula no se necesita que el paciente o un asistente halen la lengua.

4) Se describe la mejor manera de construir el espejo-cánula.

# Sociedad Argentina de Tisiologia

RODOLFO E. CUCCHIANI ACEVEDO, M.D.
Secretary

The Executive Committee of the Sociedad Argentina de Tisiologica appointed Dr. Oscar P. Aguilar, Dr. Juan R. Paso, and Dr. Rodolfo Cucchiani Acevedo as a permanent committee to investigate the use of streptomycin in tuberculosis, and the following statement is made as a result of their study.

"Special sessions of the Sociedad Argentina de Tisiologia, held on October 23, and 24, 1947, were devoted to the question of streptomycin in the treatment of tuberculosis. The 'clinical protocol' presented at this meeting and the results published in the Primer Congreso Argentino de Tisiologia agree with the experience of the men in other countries, especially the United States. The results of this investigation are as follows:

1) Streptomycin represents a real advance in the treatment of tuberculosis; however, the effectiveness of this drug is variable and depends upon such factors as the type of disease, the stage of activity, whether it is a pulmonary or an extrapulmonary disease.

2) The use of this drug is too recent to evaluate properly, from the standpoint of lasting effect, the results obtained from streptomycin therapy.

3) The results obtained in the treatment of tuberculous meningitis are sufficiently promising to advocate the early and intensive use of streptomycin by the intramuscular and intrathecal method in all patients in whom the clinical diagnosis of tuberculous meningitis have been made.

4) Streptomycin should also be used parenterally in cases of acute miliary tuberculosis and in cases of hematogenous dissemination that precedes chronic tuberculosis with foci in various organs.

5) Streptomycin is effective in the treatment of fistulae of tuberculous origin.

6) Results obtained from streptomycin therapy in tuberculous lesions of the larynx, bronchus, or trachea are encouraging. Similar results have been observed in patients with ulcerative lesions of the digestive tract.

7) Use of streptomycin is justified in the acute forms of pulmonary tuberculosis. However, this is not always the case in

pneumonic or bronchopneumonic episodes that occur in advanced cases of chronic pulmonary tuberculosis.

8) Streptomycin is not effective in the treatment of fibrous or fibrocaseous chronic pulmonary tuberculosis and should not be used in such cases. The use of streptomycin is also contraindicated in early cases of tuberculosis that are amenable to other, recognized forms of therapy because of the toxicity that accompanies the use of this drug.

9) In general, streptomycin is an adjunct to, and does not take the place of therapeutic measures of proved effectiveness in pulmonary tuberculosis. Thus, it may be considered as an adjunct to collapse therapy, particularly when an inflammation of the bronchus interferes with cavity closure.

10) It is effective as a preventive and curative measure in postoperative tuberculous complications.

11) Streptomycin seems to be destined to expand the range of indications for collapse therapy and surgical treatment of tuberculosis.

12) It is advisable that streptomycin should be used under the control of the phthisiologist as much as possible."

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## CORRECTION IN THE JULY-AUGUST ISSUE

In Dr. A. J. Steiner's Discussion of Dr. C. C. Macklin's article, "Respiratory Volume Changes in the Pulmonary Blood Vessels in Relation to Artificial Relaxation Therapy," on page 548; the percentages given were incorrectly printed. It should have read, "The areas of two pulmonary arteries increased on inspiration; a large one increased 35 per cent, and a smaller one increased 40 per cent."

# EDITORIAL

## "Diseases of the Chest" Now Published Monthly

With this issue *Diseases of the Chest* becomes a monthly publication. During the past few years the demand of authors for the publication of their papers became so great that a large backlog of manuscript accumulated. Not infrequently papers were published almost two years after they were accepted. Moreover, many good papers were returned to their authors because of lack of space in the Journal. This posed a serious and embarrassing problem for the Editorial Board. Although the number of pages was increased from 610 in 1946 to 750 in 1947, and 950 in 1948, the demand continued to far exceed our ability to publish within a reasonable time. Therefore, on June 17, 1948 the Editorial Board proposed that a serious effort be made to arrange for monthly publication of the Journal, beginning with the January, 1949 issue. The Board of Regents carefully considered this proposal and voted favorably on June 20th.

In the future, as in the past, every effort will be made to choose papers on the basis of such factors as timeliness, presentation of new facts, procedures, etc. The return of a manuscript to its author will not necessarily mean that it is unworthy of publication; it may be rejected because other papers have been accepted or recently published on the same or closely related subjects, etc. It is desirable to produce a well balanced journal.

*Diseases of the Chest* is the medium through which members of the American College of Chest Physicians and others throughout the world present the results of their investigations to physicians and scientists everywhere. It is the medium through which diagnostic, therapeutic and preventive measures are presented and evaluated, and through which members of the numerous College Chapters and chest physicians throughout the world keep in touch with one another. Thus they are knitted into one large family, each member of which takes pride in the accomplishment of the others, and all are striving to relieve the sick and maintain good health among the peoples of the world, whoever they are and wherever they may be.

In its endeavor to make *Diseases of the Chest* a good medium, the Editorial Board has had the unqualified support and confidence of the members of the College and many others. With more space now available for communications in the Journal, they are desirous of not only maintaining previous standards but also elevating them with every opportunity. To this end the Editorial Board solicits criticism, suggestions and advice from physicians everywhere.

J. A. M.

## POSTGRADUATE COURSE IN DISEASES OF THE CHEST, AMERICAN COLLEGE OF CHEST PHYSICIANS

November 8-13, 1948, Hotel New Yorker, New York City

Some of the physicians and instructors who participated in the Postgraduate Course in Diseases of the Chest of the American College of Chest Physicians.

## Fifteenth Annual Meeting American College of Chest Physicians

The Fifteenth Annual Meeting of the American College of Chest Physicians will be held at the Ambassador Hotel, Atlantic City, New Jersey, June 2 through 5, 1949, just prior to the annual meeting of the American Medical Association, June 6 through 10. Members are urged to make their hotel reservations at once by writing to the Executive Offices of the College, 500 North Dearborn Street, Chicago 10, Illinois, giving their arrival and departure dates as well as the type of accommodations desired. A limited number of rooms are available at the Ambassador Hotel for members wishing to remain for both meetings.

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## Second Annual Postgraduate Course in Diseases of the Chest to be Given in Philadelphia

The Second Annual Postgraduate Course in Diseases of the Chest sponsored by the Council on Postgraduate Medical Education and the Pennsylvania Chapter of the American College of Chest Physicians and the Laennec Society of Philadelphia will be presented during the week of February 28 - March 5, at the Warwick Hotel, Philadelphia, Penna.

Tuition fee for the course is $50.00 and registration, although limited in number, is open to all physicians. Application for the course may be made to the American College of Chest Physicians, 500 North Dearborn Street, Chicago 10, Illinois. Applications will be accepted in the order in which they are received. See front advertising page vi for application form.

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## VIII Congreso Pan-Americano de la Tuberculosis (ULAST)

### *Mexico City, Mexico*

**JANUARY 23 - 29, 1949**

The following are the main themes of ULAST:

1) Influencia del Trabajo en el origen y desarrollo de la tuberculosis.
2) Balance de la Terapéutica quirúrgica en la Tuberculosis pulmonar.
3) Estudios sobre la función cardio respiratorio en la tuberculosis.

### THIRD NATIONAL CONGRESS OF TUBERCULOSIS AND SILICOSIS

In conjunction with ULAST, the Third National Congress of Tuberculosis and Silicosis will take place in the National Institute of Cardiology, Mexico City, Mexico, during January 23-29, 1949. The following are the officials of the Congress:

President, Dr. Alejandro Celis; Vice-President, Dr. Juan B. Meana; Secretary, Dr. Ermilo Esquivel. The Honorary Presidents are as follows: Lic. Miguel Alemán, President of the Republic of Mexico; Dr. Rafael P. Gamboa, Secretary of Public Health; and Lic. Manuel Ramírez Vásquez, Secretary of Labor.

*Sections of the Third National Congress of Tuberculosis and Silicosis*

1) Epidemiología y Profilaxis de la Tuberculosis.
   Pres.: Dr. Manuel Beltrán del Río.
   Sec.: Dr. Rafael Sentíes.
2) Colapso gaseoso y tratamiento médico de la Tuberculosis pulmonar.
   Pres.: Dr. Manuel Nava.
   Sec.: Dr. José Ramírez Cano.
3) Tratamiento quirúrgico de la Tuberculosis Pulmonar.
   Pres.: Dr. Alfonso Aldama.
   Sec.: Dr. Rodolfo Gil.
4) Localizaciones extrapulmonares de la Tuberculosis.
   Pres.: Dr. Alejandro Castanedo.
   Sec.: Dr. Javier Lomelí, Dr. Ernesto Escalona, Dr. Arturo Novoa.
5) Padecimientos Respiratorios no Tuberculosos.
   Pres.: Dr. Manuel Díaz Estúa.
   Sec.: Dr. Carlos Jiménez Caballero.
6) Neumoconiosis.
   Pres.: Dr. Jorge Karam.
   Sec.: Dr. Javier García Luna.
7) Anatomía Patológica y Laboratorio de la Tuberculosis Pulmonar.
   Pres.: Dr. Arsenio Gómez Muriel.
   Sec.: Dr. Maximiliano Salas.
8) Clínica y Radiología de la Tuberculosis Pulmonar.
   Pres.: Dr. Horacio Rubio Palacios.
   Sec.: Dr. Rogelio Bolaños.

The Executive Offices of the College will be pleased to assist in arrangements for those members who desire to attend ULAST and the Third National Congress of Tuberculosis and Silicosis.

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## COUNCIL ON RESEARCH

Charles M. Hendricks, M.D., El Paso, Texas, General Chairman

*Scientific Section:*

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J. Winthrop Peabody, M.D., Washington, D. C.
J. C. Placak, M.D., Cleveland, Ohio
Paul A. Turner, M.D., Louisville, Kentucky

# College Chapter News

## CUBAN CHAPTER

The Cuban Chapter of the College met in Havana on November 25 and elected the following officers for the coming year:

Francisco J. Menendez, M.D., Havana, President
R. Sanchez Acosta, M.D., Havana, Vice-President
Carlos Barroso, M.D., Havana, Secretary-Treasurer

The meeting was attended by Dr. Octavio Rivero, Governor; Dr. Gustavo Aldereguia; Dr. Francisco J. Menendez; Dr. Alfredo Antonetti; Dr. Orfilio Suarez Bustamente; Dr. R. Sanchez Acosta; Dr. Rene G. Mendoza; and Dr. Antonio Navarrete, Regent of the College for Cuba.

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## ILLINOIS CHAPTER

The Illinois Chapter of the College sponsored a dinner and scientific meeting at the Congress Hotel, Chicago, on Friday evening, December 10. A symposium on "Problems in the Management of Asthma" was presented in which the following physicians took part:

"Allergic Aspects," Leon Unger, M.D., F.C.C.P., Chicago.
"Psychosomatic Aspects," George C. Hamm, M.D., Chicago.
"Physiologic Aspects," Edwin R. Levine, M.D., F.C.C.P., Chicago.

The program was followed by an open discussion in which all physicians present participated.

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## MEXICAN CHAPTER

The annual meeting of the Mexican Chapter was held in Mexico City on December 1, 1948. The following officials were elected to serve in 1949:

Ismael Cosio Villegas, M.D., Mexico City, President
Miguel Jimenez, M.D., Mexico City, Vice-President
Horacio Rubio Palacios, M.D., Mexico City, Secretary-Treasurer

A committee for the study of the By-Laws of the Mexican Chapter was appointed. Dr. Manuel Alonso and Dr. Miguel Jimenez will comprise the committee.

A Board of Examiners, consisting of Dr. Fernando Rebora and Dr. Carlos Noble, has been organized to pass upon all applicants applying for Fellowship in the College.

The chapter resolved to lend its fullest support to the VIII Pan American Congress on Tuberculosis which is being held in Mexico City, January 23-29, 1949.

CUBAN CHAPTER MEETING, SCIENTIFIC ACADEMY, HAVANA, CUBA, OCTOBER 26, 1948

*At Speaker's Table:* Antonio Navarrete, M.D., F.C.C.P., Havana, Regent of the College for Cuba; (standing) Rene G. Mendoza, M.D., Havana, Chairman of the Program Committee, Cuban Chapter; Louis Mark, M.D., F.C.C.P., Columbus, Ohio, First Vice-President of the College; Octavio Rivero, M.D., Havana, Governor of the College for Cuba. Seated at left, background: Mr. Murray Kornfeld, Chicago, Illinois, Executive Secretary of the College; Juan R. Herradora, M.D., F.C.C.P., Jersey City, New Jersey, Secretary, Council on Pan American Affairs; and Edgar Davis, M.D., F.C.C.P., Washington, D. C., member, Council on Pan American Affairs of the College.

### PHILIPPINE CHAPTER ORGANIZED

On November 21 the Philippine Chapter of the College was organized in Manila. Gumersindo Sayago, M.D., F.C.C.P., Cordoba, Argentina, Regent of the College for Argentina, who was visiting in the Philippine Islands on a Medical Mission sponsored by the World Health Organization and the Unitarian Service Committee, participated in the organizational meeting. The following officers of the 36th College chapter were elected:

Miguel Cañizares, M.D., Manila, President
Wenceslao Vitug, M.D., Manila, Vice-President
Fidel R. Nepomuceno, M.D., Manila, Secretary-Treasurer
Jose Avellana, M.D., Manila, Counsellor.

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### WISCONSIN CHAPTER

The Wisconsin Chapter of the College sponsored a program on diseases of the chest before the Vernon County Medical Society which met at Westby on Friday, August 27, 1948. The program presented was as follows:

"The Modern Treatment of Cough,"
Andrew L. Banyai, M.D., F.C.C.P., Milwaukee, Wisconsin.

"Diagnosis of Diseases of the Chest,"
Leon H. Hirsh, M.D., F.C.C.P., Milwaukee, Wisconsin.

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## College News Notes

Ross K. Childerhose, M.D., F.C.C.P., Harrisburg, Pennsylvania, presented a lecture on the "Principles and Treatment of Pulmonary Tuberculosis" by invitation to the members of La Société de Phtisiologie de Montréal, Canada, on Tuesday, October 26, 1948.

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The Metropolitan Washington Tuberculosis Conference for 1948 convened at Glenn Dale Sanatorium, October 28. College members who participated in the program were Brian B. Blades, M.D., F.C.C.P.; Charles P. Cake, M.D., F.C.C.P.; Daniel L. Finucane, M.D., F.C.C.P.; and A. Barklie Coulter, M.D., F.C.C.P.

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Leon H. Hetherington, M.D., F.C.C.P., Assistant Professor of Medicine at the University of Pittsburgh School of Medicine, has been appointed chief of the division of tuberculosis services of the Maryland State Department of Health. Dr. Hetherington will take over direction of Maryland's four tuberculosis sanatoriums.

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Sir Alexander Fleming, Honorary Fellow of the College, attended a meeting of the Medical Society of London on October 25, for a discussion on streptomycin. Dr. Jenner Hoskin presided. Sir Alexander Fleming stated "Certain results indicated that given by the mouth, streptomycin

had a rapid effect in infantile diarrhoea. It was a local action; no big dose need be given. This might turn out to be a very important use for this drug."

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Juan R. Herradora, M.D., F.C.C.P., Jersey City, New Jersey, Secretary of the Council on Pan American Affairs of the College, was invited to visit Guatemala as a guest of that country. Dr. Herradora visited Guatemala City during the latter part of October. Upon arrival he was greeted by the President of Guatemala and the Secretary of Public Health.

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At the request of the Philippine Government, the World Health Organization, Interim Commission, and the Unitarian Service Committee jointly sponsored a Medical Mission to the Philippine Islands to give lectures and conduct demonstrations on recent advances in certain fields of medical science as indicated by the Philippine Government. The Mission consisted of five members, including Gumersindo Sayago, M.D., F.C.C.P., Cordoba, Argentina, Regent of the College for Argentina. Before returning to his home in Cordoba, Dr. Sayago is visiting in the United States.

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On August 16th, the sixth session of the Australasian Medical Congress (British Medical Association) was formally opened in Perth. W. B. Cotter Harvey, M.D., F.C.C.P., was President of the Section on Public Health, Tuberculosis and Tropical Medicine. On August 20th, a public meeting was held at which the first steps were taken to form an Australian Tuberculosis Association. Among officers elected to the Association were the following College members: Dr. R. W. Cowan, M.D., F.C.C.P., Adelaide; W. B. Cotter Harvey, M.D., F.C.C.P., Sydney; and Sir Sidney Sewell, F.C.C.P., Melbourne.

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## BACK ISSUES OF THE JOURNAL IN DEMAND

The following back issues of the journal are in demand. One dollar will be paid for each of the issues listed below upon receipt at the executive offices of the American College of Chest Physicians in Chicago.

Volume IX, Nos. 1, 2, 3, 4 (1943)
Volume X, Nos. 1, 2, 3, 4, 6 (1944)
Volume XI, Nos. 1, 2, 3 (1945)
Volume XII, Nos. 1, 2, 3, 4, 5, 6 (1946)
Volume XIII, Nos. 1, 2 (1947)
Volume XIV, No. 1 (1948)

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## POSTGRADUATE COURSE IN BRONCHOESOPHAGOLOGY

The University of Illinois, Department of Otolaryngology, will offer a two week Bronchoesophagology Course under the direction of Paul H. Holinger, M.D., F.C.C.P., from February 7 through February 19, 1949. The tuition and laboratory fee is $150.00 and applications can be obtained by writing to the University of Illinois, Department of Otolaryngology, 1853 West Polk Street, Chicago 12, Illinois.

## TWENTY-FIRST ANNIVERSARY YEAR OF HAROFE HAIVRI

The appearance of Volume I—1948 of the *Harofe Haivri*, The Hebrew Medical Journal, inaugurates the 21st successful year of its publication under the editorship of Moses Einhorn, M.D. The Journal's contents are not confined to technical medical topics but is divided into several sections covering a variety of related subjects of interest to the medical profession.

The original articles are summarized in English to make them available to those who are unable to read Hebrew. For further information, communicate with the editorial office of the Hebrew Medical Journal, 983 Park Avenue, New York 28, New York.

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## ANNUAL SESSION, AMERICAN COLLEGE OF PHYSICIANS

The American College of Physicians will conduct its 30th Annual Session at New York, N. Y., March 28 through April 1, 1949. Dr. Franklin M. Hanger, Jr., of New York City is the Chairman for local arrangements and the program of Clinics and Panel Discussions. The President of the College, Dr. Walter W. Palmer, Director of The Public Health Research Institute of the City of New York, Inc., and Professor Emeritus, Columbia University College of Physicians and Surgeons, is in charge of the program of Morning Lectures and afternoon General Sessions.

Secretaries of medical societies are especially asked to note these dates and, in arranging meeting dates of their societies, to avoid conflicts with the College Meeting, for obvious mutual benefits.

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## BINDING FOR COMPLETED VOLUMES

We are pleased to announce that The Book Shop Bindery, 308 West Randolph Street, Chicago, Illinois, will produce a well-bound volume at as low a price as possible for those members and subscribers who wish to preserve their issues of "Diseases of the Chest." They will bind the six issues of Volume 14 in the best grade of washable buckram with gold stamping on the spine and the member's or subscriber's name in gold on the front cover. Please send the six issues to Chicago by express or parcel post prepaid with check or money order for $2.75 made payable to The Book Shop Bindery. The bound volume will be returned with transportation prepaid by the bindery.

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# Book Reviews

*Zinsser's Textbook of Bacteriology*. By David Smith and Donald S. Martin, et al, Duke University School of Medicine. Cloth. Price $10.00. Pp. 992, Illustrated. Appleton Century Crofts, New York, 1948.

In 1935 there appeared, from the pen of Hans Zinsser, a book "Rats, Lice and History" which was the life history of typhus fever. In this book there was a lucid, simple and fascinating story of the effect of infections on, and the transmission of infectious diseases to the population, and the impetus it had on civilization. In 1940 Zinsser's last publication, "As I Remember Him," appeared. This, the reader will recall, was in essence an autobiography of Hans Zinsser. From these books one readily concludes that here was an author of first magnitude, a scientist of international reputation, and an authority in his particular field —bacteriology and public health.

For many years before the above mentioned books appeared, Zinsser's contribution to bacteriology was known to every medical student through his texts, first, with Professor Hiss (published in 1910), later published under his name alone, and finally in cooperation with Bayne-Jones. For some years thereafter, this book did not appear on the market and was not available to the medical student, but due to the efforts of Smith and Martin and their associates, the ninth edition of Zinsser's Textbook of Bacteriology is now available.

Smith, Martin and their associates are to be complimented for their work in revising and re-editing Zinsser's Textbook of Bacteriology, which is generally accepted as a classic in this particular field. There is the added advantage that Smith, Martin, et al, not only have had the benefit of Zinsser's Textbook of Bacteriology as students, but have also had the advantage of teaching bacteriology to their own students, and have thus been able to benefit from their wide experience in bringing this book up to date.

These new authors have introduced each subject with the public health aspect of the disease. They have further added the newer investigations in the field of bacteriology and public health which accummulated during and after World War II. Since the advent of bacteriologic and cultural characteristics of organisms, emphasis has been placed in various chapters of the book so that this newer knowledge shall not escape detection by the reader. Zinsser, among other phases of his work, laid particular emphasis on immunology. The new authors have not only rewritten and brought up to date this subject within the realms of the function of their textbook, but have also rewritten the material concerning bacteriological metabolism, fungi, and virus diseases.

We can illustrate the format of this book with the subject of tuberculosis. In this chapter there is a brief resumé of the public health aspects of tuberculosis, followed by discussion of the genus *Myobacterium* found in the soil, water and air. In a clear, simple and understandable manner, there is a discussion of the cultural characteristics as well as the staining properties of the organism; tuberculin and its various products in diagnostic procedures; the effect of the antibiotics on the organism; and experimental methods for the detection of the disease. The subject is further integrated by a brief clinical resumé of tuberculosis and finally the public health aspects of the subject. This method of reviewing the bacteriologic, cultural, public health and clin-

ical aspects of the disease is indeed helpful to the student. Included is a chapter dealing with technical methods of bacteriology, immunology, and serology which is ample for the medical student.

This book can be unqualifiedly recommended to the medical student and the practicing physician as a source of information in bacteriology. The publishers are to be complimented on the manner in which the book has been printed and the clear photographs that appear in the text. Although the book has been designated as a "Textbook of Bacteriology," it should be in the possession of medical students and practitioners of medicine and public health as a guide in the application of bacteriology and immunology to diagnosis, therapy, and prevention of infectious diseases.

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*A Practical Manual of Diseases of the Chest.* By Maurice Davidson, M.A., M.D., F.R.C.P. Third Edition, 1948. Oxford University Press.

This extensive manual, comprising six hundred and seventy pages, has been written in the best literary style and diction, and reads like a story book. The author has presented a well systematized sequence of his subject matter, with appropriate illustrations and photographs of x-ray films, as well as clinical charts. He has made free use of illustrative case histories to emphasize the discussion of each subject. The more recent advances in the chemotherapy and antibiotic therapy of pneumonias have been thoroughly discussed. It has considerable material of interest to the internist, as well as the thoracic surgeon, to make the book worthy of a place in their library. Although the book is primarily written for the British public its international scope should make it one of choice for everyone interested in diseases of the chest.

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## BOOKS, REPORTS AND REPRINTS RECEIVED

"The Relationship of Tuberculosis and Silicosis," O. A. Sander, M.D., F.A.C.P., Milwaukee, Wisconsin.

"Benign Pneumoconiosis Due to Metal Fumes and Dusts," O. A. Sander, M.D., F.A.C.P., Milwaukee, Wisconsin.

"Relacoes Entre a Tuberculose Pulmonar e a Tuberculose Osteo-Articular," Lopo de Carvalho Cancella, Lisbon, Portugal.

"Micro-radiofotografia em massa; resultados obtidos numa primeira observacao," Lopo de Carvalho Cancella, Lisbon, Portugal.

"A Posicao de Drenagem e a Penicilina Por Via Inalatoria Como Tempo Pre-operatorio da Cirurgia Pulmonar," Lopo de Carvalho Cancella, e F. Ferreira Coelho, Lisbon, Portugal.

"Tratemento do Empiema Pleural Tuberculoso," Dr. Jesse Teixeira, Rio de Janeiro, Brazil.

"Die primare Tuberkulose bei Erwachsenen und Kindern und ihre Entwicklung," Dozent Dr. Med. St. J. Leitner, published by Medizinischer Verlag Hans Huber, Bern, Switzerland.

"Extrapleural Pneumonolysis with Lucite Plombage," Drs. John B. Grow and Ralph E. Dwork, Denver, British Journal of Tuberculosis and Diseases of the Chest, April 1948.

'Colloidal Copper Morrhuate," Dr. Paulo Seabra, Rio de Janeiro, Brazil.

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# Obituary

## PIERRE AMEUILLE

1880 - 1947

Dr. Pierre Ameuille was born in 1880 in Nevers, France, and died at his home in Paris on December 19, 1947. He went to Paris to study medicine and graduated from medical school there. Dr. Ameuille completed his internship at the "Hopitaux de Paris." His interests during the following period were particularly concerned with pathology and anatomic research. During the first World War, in cooperation with English medical officers, he studied many cases of war nephritis and antityphic vaccination.

Dr. Ameuille was associated with the "Hopitaux de Paris" and the "Hopital Cochin" where he specialized in pulmonary diseases, tuberculosis, bronchial pathology and physio-pathology. He was President of the "Societe Medicale des Hopitaux de Paris," a member of the "Academie de Medicine," and a member of the American College of Chest Physicians, as well as a number of other societies. Just prior to his death he organized and directed the "Societe Francaise de Pathologie Respiratoire." His knowledge and intelligence, his philosophical outlook, his serenity in spite of troubles, made Dr. Ameuille one of the greatest physicians in France.

J. M. Lemoine, M.D., Paris, France.

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## SIX PHYSICIANS WINNERS OF ANNUAL LIPIODOL AWARDS FOR 1948

Physicians from all sections of the country submitted examples of diagnostic roentgenography in the Annual Lipiodol Awards program for 1948. The technical excellence of the many entries offer further evidence of the great progress achieved in this specialty.

Awards were made to physicians in four categories of roentgenography. Entries submitted were judged for the extent of diagnostic features revealed and general technical excellence of plates in otolaryngology, bronchography, utero-salpingography and the delineation of sinus tracts. Physicians receiving awards were: otolaryngology, Dr. Henry M. Scheer, New York; bronchography, Dr. J. A. Crellin, Philadelphia, Fellow of the American College of Chest Physicians, and Dr. Louis Schneider, New York; utero-salpingography, Dr. A. P. Echternacht, Crawfordsville, Indiana, and Dr. B. S. Epstein, New York; sinus tracts, Dr. Sidney E. Foster, San Francisco, and Dr. B. S. Epstein, New York.

Entries were reviewed by a distinguished board of judges consisting of Dr. Lawrence Reynolds, President of the American Roentgen Ray Society; Dr. Ira H. Lockwood of Kansas City, Missouri; Dr. Irving Schwartz of New York.